Interim Session, American College of Chest Physicians
Denver, Colorado, November 30, December 1, 1952

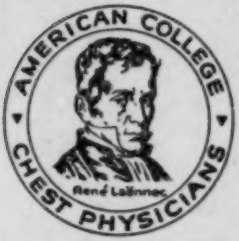
VOLUME XXII NUMBER 2

DISEASES

of the

CHEST

OFFICIAL PUBLICATION



PUBLISHED MONTHLY

AUGUST
1952

EXECUTIVE OFFICE, 112 EAST CHESTNUT STREET, CHICAGO 11, ILLINOIS
PUBLICATION OFFICE, ALAMOGORDO ROAD, EL PASO, TEXAS

Entered as Second Class Matter August 18, 1936, at the Postoffice at El Paso, Texas
Under the Act of Congress of August 12, 1912.
Copyright, 1952, by the American College of Chest Physicians

XII Congress, International Union Against Tuberculosis
II International Congress on Diseases of the Chest
Rio de Janeiro, Brazil, August 24-30, 1952

JACKSON BRONCHOSCOPES

by Pilling

Pilling supplies the complete line of genuine Jackson bronchoscopes . . . *the approved models*, standard for use with all equipment in the standard Jackson-type Clinic.

Pilling makes *every size* and *every type* of bronchoscope devised by Dr. Chevalier Jackson, and makes them—for years of service—from Broncalloy.

Broncalloy instruments are lighter, yet stronger—thin but rigid—smooth in surface, bite-proof and resistant to corrosion, distortion and damage.

Standard Jackson Bronchoscopes, as illustrated, all sizes from 4 mm. x 20 cm. to 9 mm. x 40 cm., with 2 light carriers and 2 lamps. Light carriers are interchangeable in bronchoscopes of equal length.

Special sizes can be made to order. Consult us.

Order direct from

George P. Pilling and Son Company

3451 WALNUT STREET • PHILADELPHIA

Headquarters for

INSTRUMENTS FOR BRONCHOESOPHAGOLOGY

A Standing Invitation. When in Philadelphia, visit our new salesroom. Free parking for doctors on our private lot.

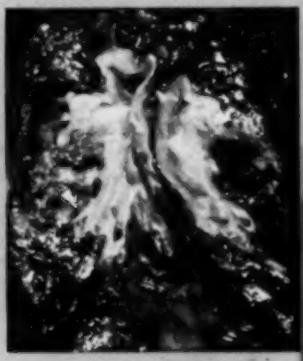
Record it

... with photographs in black and white, or color

HOSPITALS and clinics, physicians and surgeons, more and more of them, are *making photography routine.* As a result, case histories are more accurate, more comprehensive, less bulky; files are full of "live" material for teaching, diagnosis, research, reference.

Carcinoma of bronchus.

Adenoma of bronchus.



Squamous carcinoma of bronchus.

Record it

... with the Kodak Master View Camera 4 x 5

GET top-quality medical photographs with this compact, lightweight view camera. Combines great structural rigidity with operating flexibility. Has revolving back, rising-falling front, horizontal and vertical swings. Wide choice of Ektar lenses—all color-corrected—all with glass-air surfaces Lumenized. List price—camera, carrying case, holder—$145, subject to change without notice. Lenses extra.

For further information, see your photographic dealer or write:

EASTMAN KODAK COMPANY
Medical Division, Rochester 4, N. Y.

Complete line of Kodak Photographic Products for the Medical Profession includes: cameras and projectors—still- and motion-picture; film—full color and black-and-white (including infrared); papers; processing chemicals; microfilming equipment and microfilm.

Serving medical progress through Photography and Radiography

Kodak
TRADE-MARK

SEVENTH ANNUAL POSTGRADUATE COURSE IN THE RECENT ADVANCES IN DISEASES OF THE CHEST

Sponsored by the

COUNCIL ON POSTGRADUATE MEDICAL EDUCATION

of the

AMERICAN COLLEGE OF CHEST PHYSICIANS

In Cooperation With The

ILLINOIS STATE CHAPTER

and the

Faculties of the Medical Schools and Hospitals of Chicago

HOTEL ST. CLAIR : CHICAGO, ILLINOIS

OCTOBER 6 - 10, 1952 : TUITION: $50.00

— Registration Will Be Limited —

AMERICAN COLLEGE OF CHEST PHYSICIANS
112 East Chestnut Street, Chicago 11, Illinois

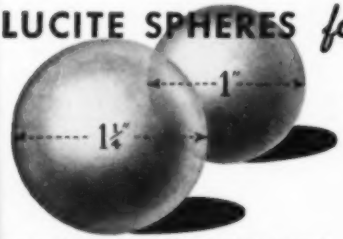
Enclosed you will please find my registration fee in the amount of $50.00 for the Seventh Annual Postgraduate Course in Diseases of the Chest to be held in Chicago, Illinois.

NAME .....

ADDRESS .....

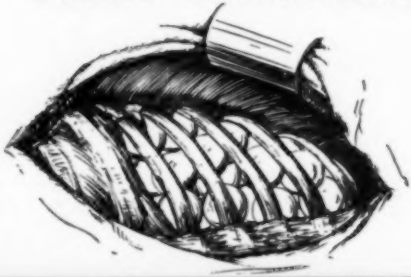
CITY & STATE .....

LUCITE SPHERES *for Plumbage Thoracoplasty*



. . . as used by the

OVERHOLT THORACIC CLINIC



Lucite Spheres, 1¼" or 1" (in diam.) . . . $.30 ea.

Polyethylene Film, Animal Tested, (Used for encasing Spheres)

54" x .015 . . . 10 yds. $7.50
54" x .002 . . . 10 yds. $7.50

ORDER DIRECT FROM:

CJ
ESTABLISHED 1838

Codman & Shurtleff, Inc.

MAKERS OF SURGEONS' INSTRUMENTS

104 BROOKLINE AVENUE BOSTON 15, MASSACHUSETTS

DISEASES *of the* CHEST

OFFICIAL PUBLICATION

OF THE

AMERICAN COLLEGE OF CHEST PHYSICIANS

EDITORIAL BOARD

JAY ARTHUR MYERS, M.D., Chairman
Minneapolis, Minnesota
Editor-in-Chief

CHARLES M. HENDRICKS, M.D.
El Paso, Texas
Editor Emeritus

MILTON W. ANDERSON, M.D.
Rochester, Minnesota

RICHARD H. OVERHOLT, M.D.
Brookline, Massachusetts

ANDREW L. BANYAI, M.D.
Milwaukee, Wisconsin

HENRY C. SWEANY, M.D.
Jacksonville, Florida

ASSOCIATE EDITORS

ANTONIO A. ADAMES, M.D.....Holtville, California
WILLIAM B. BEAN, M.D.....Iowa City, Iowa
EDWARD P. EGLEE, M.D.....New York, New York
SEYMOUR M. FARBER, M.D.....San Francisco, California
EDWARD W. HAYES, M.D.....Monrovia, California
HANS H. HECHT, M.D.....Salt Lake City, Utah
PAUL H. HOLINGER, M.D.....Chicago, Illinois
CHEVALIER L. JACKSON, M.D.....Philadelphia, Pennsylvania
HOLLIS E. JOHNSON, M.D.....Nashville, Tennessee
ARTHUR M. MASTER, M.D.....New York, New York
EDGAR MAYER, M.D.....New York, New York
ALTON OCHSNER, M.D.....New Orleans, Louisiana
GEORGE G. ORNSTEIN, M.D.....New York, New York
J. WINTHROP PEABODY, M.D.....Washington, D. C.
ARTHUR Q. PENTA, M.D.....Schenectady, New York
LEO G. RIGLER, M.D.....Minneapolis, Minnesota

CORRESPONDING ASSOCIATE EDITORS

Donato G. Alarcon, M.D., Mexico
Adrian Anglin, M.D., Canada
Jose Ignacio Baldo, M.D., Venezuela
Etienne Bernard, M.D., France
Miguel Canizares, M.D., Philippine Is.
Manoel de Abreu, M.D., Brazil
Lopo de Carvalho, M.D., Portugal
Sir Alexander Fleming, England
Ovidio Garcia Rosell, M.D., Peru
Fernando D. Gomez, M.D., Uruguay
Affonso MacDowell, M.D., Brazil
David P. Marais, M.D., South Africa
Amadeo V. Mastellari, M.D., Panama
Gustav Maurer, M.D., Switzerland
Andre Meyer, M.D., France
Papken S. Mugriditchian, M.D., Lebanon
Antonio Navarrete, M.D., Cuba
Juda M. Pauzner, M.D., Israel
Hector Orrego Puelma, M.D., Chile
Raul F. Vaccarezza, M.D., Argentina
Raman Viswanathan, M.D., India
Harry W. Wunderly, M.D., Australia
Attilio Omodei Zorini, M.D., Italy

EXECUTIVE OFFICE
112 East Chestnut Street, Chicago 11, Illinois
MURRAY KORNFELD, *Managing Editor*

VOLUME XXII AUGUST 1952 NUMBER 2

CONTENTS:

A SIMPLE METHOD FOR THE DETERMINATION OF VITAL CAPACITY - TIME RELATIONSHIPS 123
Maurice S. Segal, M.D., J. Aaron Herschfus, M.D. and Mauricio J. Dulfano, M.D., Boston, Massachusetts

VENOUS PRESSURE AS A GUIDE TO PNEUMOPERITONEUM THERAPY IN PULMONARY EMPHYSEMA 130
Gustav J. Beck, M.D., A. Chesmore Eastlake, Jr., M.D. and Alvan L. Barach, M.D., New York, New York
Discussion: Leon H. Hirsh, M.D., Milwaukee, Wisconsin

PHYSIOLOGIC FACTORS IN THE USE OF THE BODY RESPIRATOR FOR IMPAIRED RESPIRATORY FUNCTION 141
James L. Whittenberger, M.D. and James V. Maloney, Jr., M.D., Boston, Massachusetts
Discussion: Alvan L. Barach, M.D., New York, New York

THE SURGICAL TREATMENT OF PULMONIC VALVULAR STENOSIS 152
William H. Muller, Jr., M.D., Los Angeles, California

BOECK'S SARCOID: OBSERVATIONS ON THE USE OF BCG VACCINE 159
Marvin S. Harris, M.D. and Charles Shore, M.D., Los Angeles, California

DELAYED REACTIONS TO BOUILLON MEDIUM OF TUBERCULIN 163
M. R. Lichtenstein, M.D. and C. A. Cilella, M.D., Chicago, Illinois
Discussion: E. A. Piszek, M.D., Chicago, Illinois

LINGULAR DISEASE 171
William A. Hopkins, M.D. and Ted F. Leigh, M.D., Atlanta, Georgia

MYASTHENIA GRAVIS, DIABETES MELLITUS AND INTRAVENTRICULAR BLOCK, AS CO-EXISTENT COMPLICATIONS OF PULMONARY TUBERCULOSIS 183
Edward T. Schantz, M.D., San Antonio, Texas

BASAL METABOLISM IN PULMONARY TUBERCULOSIS 187
George C. Leiner, M.D. and Sol Abramowitz, Staten Island, New York

PLEURO-PNEUMONECTOMY IN THE TREATMENT OF TUBERCULOUS EMPYEMA 193
Jos Daems, M.D. and Robert Pannier, M.D., Bruges, Belgium

COUGH: ITS PATHOLOGY, THERAPY AND MANAGEMENT 196
A. Worth Hobby, M.D., Atlanta, Georgia

PULMONARY ASPERGILLOSIS IN GREECE 206
Panayotis Chortis, M.D., Athens, Greece

OBSCURED BRONCHIOGENIC CARCINOMA. REPORT OF A CASE WITH NECROPSY 213
Isaac Epstein, M.D. and Marjorie J. Williams, M. B. Ch. B., Temple, Texas

TWO CASES OF INTRA-BRONCHIAL FOREIGN BODIES OF LONG DURATION 219
Juan M. Boettner, M.D., Manuel Rivero, M.D., Miguel A. Perito, M.D., Rafael Codas, M.D., Nelson Llamosas, M.D. and Carlos Maas, M.D., Asuncion, Paraguay

SPONTANEOUS HEMOPNEUMOTHORAX: Report of Two Cases 226
William H. Kastl, M.D., Alexandria, Louisiana

EIGHTEENTH ANNUAL MEETING OF THE COLLEGE 233

Entered as second class matter, August 18, 1936, at the postoffice at El Paso, Tex., under the Act of Aug. 24, 1912

Annual Subscription: United States of America $8.50
Foreign Countries $10.00

KELEKET *Exclusive*
Ceiling-Mounted X-ray
MOTOR DRIVEN Tube Crane

Clear the floor for action with the Keleket *Exclusive* Ceiling-Mounted Tube Crane. Suspended entirely from the ceiling, it offers effortless convenience in every radiographic and therapy technic.

The Keleket Tube Crane does more than the ordinary tubestand and it conserves valuable floor space. There are no rails on the floor, no obstruction whatever to the operator's complete freedom. The layout of your radiographic facilities becomes much more flexible with the Tube Crane.

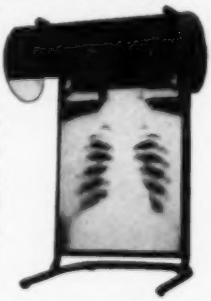
Brought to practical reality by Keleket, the Ceiling-Mounted Tube Crane offers unparalleled tube manipulation, three stereoscopic shifts, fingertip positioning and precise indication of angulation.

Write for FREE brochure

KELEKET X-RAY CORPORATION
214-8 W. 4th St., Covington, Ky.
(Kelley-Koett The Oldest Name in X-Ray)
Export Sales: Kelley-Koett International Corp.
215 E. 37th Street, New York 16, New York

POWERS X-RAY PAPER REDUCES COSTS 50% OR MORE!

For most routine work, radiographs of high quality can be made at less than half the usual cost with Powers X-Ray Paper. That is why more and more hospitals are using *both* paper and celluloid base film in their X-Ray departments. Techniques differ only slightly.



Proven in use for over 16 years, Powers X-Ray Paper comes in standard sheet sizes, or perforated rolls for use with the Powers Magazine Cassette.

Let us show you in detail how you can effect substantial savings with Powers X-Ray Paper. Write for complete information and literature.

POWERS X-RAY PRODUCTS, INC.

Reg. U.S. Pat. Off.
POWERS X-RAY PRODUCTS
Group Radiography
Glen Cove, Long Island, N. Y.

SIXTH ANNUAL POSTGRADUATE COURSE IN DISEASES OF THE CHEST

Sponsored by the

COUNCIL ON POSTGRADUATE MEDICAL EDUCATION

of the

American College of Chest Physicians

In Cooperation With the

NEW YORK STATE CHAPTER

and the

Medical Schools and Hospitals of New York City

HOTEL NEW YORKER
New York City

NOVEMBER 10 - 14, 1952

— TUITION: $50.00 —

The Most Recent Advances in the management and treatment of chest diseases will be discussed.

Registration Will Be Limited

American College of
Chest Physicians
112 East Chestnut Street
Chicago, Illinois

Enclosed you will please find my registration fee in the amount of $50.00 for the Sixth Annual Postgraduate Course in Diseases of the Chest to be held in New York City.

Name .....

Address .....

City .....

State .....

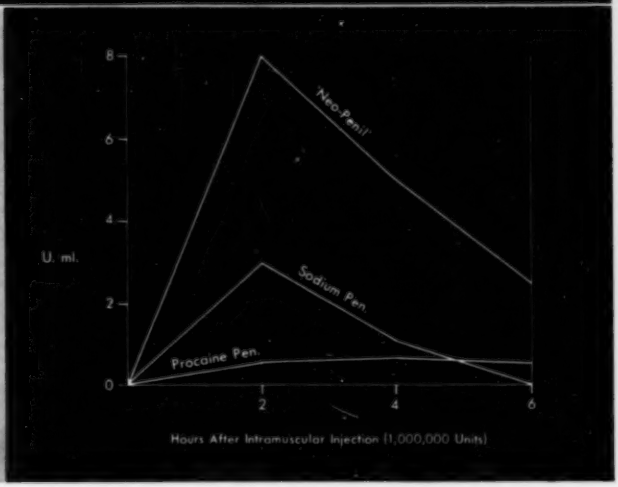
Announcing

a new chemical derivative of penicillin
which concentrates in the lung and sputum

NEO-PENIL*

for aqueous injection

Comparison of average sputum levels in humans, from Heathcote.[1]



'Neo-Penil' is a long-acting injectible penicillin, which not only assures prolonged blood levels, but also gives high concentrations in certain body tissues. For example, 'Neo-Penil' produces high concentrations *in lung tissue* and *in sputum*, and thus offers an encouraging prospect in the treatment of bronchopulmonary disease.

Indications: All infections that respond to repository penicillin.

Available: At retail pharmacies—in single-dose, silicone-treated vials of 500,000 units. Full information accompanies each vial.

Smith, Kline & French Laboratories, Philadelphia

*Trademark for penicillin G diethylaminoethyl ester hydriodide, S.K.F. Patent Applied For

1. Lancet *1*:1255 (June 9) 1951.

Now available

PYRI

(ISONICOTINIC

NEPERA

...NEPERA's new drug for the treatment of tuberculosis

Available: in scored uncoated tablets of 50 mg.—bottles of 100 and 1000.

® *Registered trademark of Nepera Chemical Co., Inc.*

PYRICIDIN®

ACID HYDRAZIDE)

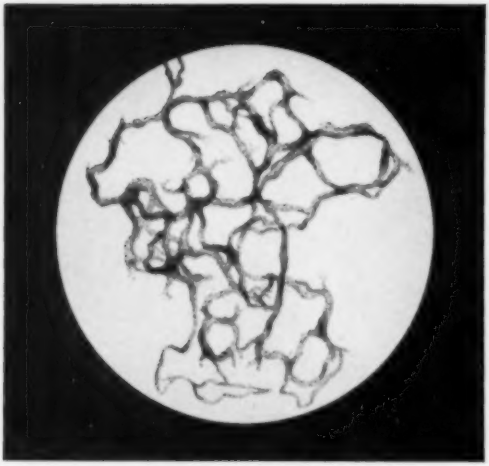
Pyricidin has provided impressive evidence of its high anti-tuberculosis efficacy, in many large-scale, long-range clinical studies; and is now available for prescription use in cases failing to respond to streptomycin therapy.

Pyricidin is manufactured by Nepera Chemical Co., Inc.—pioneers in the chemotherapy of tuberculosis, through ten years of intensive research, and specialists in the formulation of isonicotinic acid derivatives, of which Nepera is one of the largest manufacturers.

For highest quality and purity, immediate availability and economical cost, you can rely on PYRICIDIN.

Available for your prescription NOW at your local pharmacy

NEPERA CHEMICAL CO., INC.
PHARMACEUTICAL MANUFACTURERS
Nepera Park Yonkers 2, N. Y.



Serpentine growth characteristic of virulent human strains

A ready and tested measure for combating tuberculosis

As therapeutically active as streptomycin, CRYSTALLINE DIHYDROSTREPTOMYCIN SULFATE MERCK is less toxic to the vestibular apparatus, minimizes pain and swelling on injection, and may be used even in some patients allergic to streptomycin.

This preferred product is available in dry powder form and in convenient ready-to-inject form as SOLUTION OF CRYSTALLINE DIHYDROSTREPTOMYCIN SULFATE MERCK.

PARA-AMINOSALICYLIC ACID MERCK (PAS), when used in combination with CRYSTALLINE DIHYDROSTREPTOMYCIN SULFATE MERCK, prolongs the effective period of antibiotic therapy by inhibiting or delaying the development of bacterial resistance.

Crystalline Dihydrostreptomycin Sulfate Merck

Research and Production for the Nation's Health

MERCK

MERCK & CO., INC.

Manufacturing Chemists

RAHWAY, NEW JERSEY

In Canada: MERCK & CO. Limited—Montreal

© Merck & Co., Inc.

Fewer
PAS Side Actions

Pasara Calcium
Granulate

The use of Pasara Calcium Granulate has been shown to effect a significant reduction in the incidence of unfavorable side actions associated with routine oral PAS therapy. Patients who had experienced gastrointestinal discomfort with sodium para-aminosalicylate could tolerate clinically effective quantities of Pasara Calcium Granulate with virtually complete elimination of the former unfavorable reactions.[1]

DOSAGE

The average oral dose of Pasara Calcium Granulate to provide optimal blood levels is 5 Gm. administered three times daily. A single dose of this amount is therapeutically active for four to six hours.

SUPPLY

Pasara Calcium Granulate, containing 85 per cent calcium p-aminosalicylate, is available in one and 25 pound cans. It is also available in single dose packets containing 5.0 Gm. each. Pasara Calcium is also supplied in powder form in 0.5 Gm. capsules in bottles of 100 and 1,000.

The sodium-free character of Pasara Calcium makes its use particularly valuable with patients who require concurrent reduction in salt intake. The average daily requirement of sodium para-aminosalicylate, 15 Gm., yields the sodium equivalent of four grams of table salt.

Pasara Calcium Granulate, the calcium salt of para-aminosalicylic acid, is identical in clinical efficacy with that of PAS or its sodium salt.[1] When given in adequate dosage, it prevents or delays the emergence of streptomycin-resistant tubercle bacilli. It thus prolongs the period of effective antibiotic therapy.

1. Dixler, M.: Dosage and Blood Levels of Para-Aminosalicylic Acid, Schweiz. Ztschr. Tuberk. 7:6 (1950).

SMITH-DORSEY • Lincoln, Nebraska
A Division of THE WANDER COMPANY

A *Dorsey* PREPARATION

Chronic asthmatic

BACK SERVING WITH A SMILE

No need for the chronic asthmatic to give up work, play, a normal life. With NORISODRINE Sulfate, a quick-acting bronchodilating powder, symptomatic relief is as near as the patient's pocket or purse.

When the asthmatic feels an attack coming on, he simply takes three or four inhalations of the powder, using the pocket-sized AEROHALOR. Result? The bronchospasm usually ends quickly. No injections, no cumbersome equipment, no need to leave the job.

NORISODRINE is effective against both mild and severe asthma.[1,2,3] It has relatively low toxicity, and with proper administration, side-effects are few and usually minor. Before prescribing NORISODRINE, however, the physician should familiarize himself with administration, dosage and precautions. Literature may be obtained by writing Abbott Laboratories, North Chicago, Illinois. Abbott

EASY TO CARRY IN POCKET OR PURSE

NORISODRINE®

SULFATE POWDER

(ISOPROPYLARTERENOL SULFATE, ABBOTT)

for use with the AEROHALOR,® Abbott's Powder Inhaler

1. Kaufman, R., and Farmer, L. (1951), Norisodrine by Aerohalor in Asthma, Ann. Allergy, 9:89, January-February.
2. Swartz, H. (1950), Norisodrine Sulphate (25 Per Cent) Dust Inhalation in Severe Asthma, Ann. Allergy, 8:489, July-August. 3. Krasno, L., Grossman, M., and Ivy, A. (1949), The Inhalation of 1-(3',4'-Dihydroxyphenyl)-2-Isopropylaminoethanol (Norisodrine Sulfate Dust), J. Allergy, 20:111, March.

SUPPORT

in the

PNEUMOPERITONEUM TREATMENT

of

PULMONARY TUBERCULOSIS

"...A specially designed belt or corset has been utilized in all patients with any laxity of the abdominal wall. Marked distention of the abdominal wall increases the patient's discomfort and makes necessary the use of larger and more frequent refills because so much air is wasted in a larger space. Proper support will often decrease the amount of air necessary to effect an adequate rise of the diaphragms. In female patients, especially, proper support will dispose of a psychologic hazard and will help to overcome objections to the procedure..."

Edlin, James S., M.D. and Bassin, Sydney, M.D., "Pneumoperitoneum versus Pneumothorax" New York State Journal of Medicine. 50:1947 (August) 1950

THIS CAMP ABDOMINAL SUPPORT, especially designed after research and consultation with eminent authorities in the Pneumoperitoneum treatment of Pulmonary Tuberculosis, is widely prescribed. The upper contour follows the lines of the rib cage, giving firm support to the abdominal muscles without retarding breathing. Two encircling straps are used with the double back adjustments. The adjustment around the lower part of the abdomen gives firm uplift while the upper adjustment holds the support close to the body, preventing distention of the upper abdomen. Injection of air can easily be made without removing the support. The Camp adjustment is ideal for tightening the support as required by gradual absorption of air.

Write for literature.

CAMP

S. H. CAMP and COMPANY, JACKSON, MICHIGAN

World's Largest Manufacturers of Scientific Supports

Offices in New York • Chicago • Windsor, Ontario • London, England

SAY YOUR GOODBYES TO THE GI's

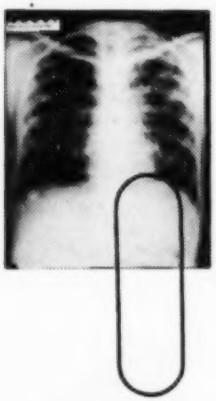
In the past, PAS therapy of tuberculosis has often been hampered by GI side reactions. Now BUFFERED PARASAL, a new, unique development of Panray, has been shown to be less irritating and less toxic than any other dosage form yet devised.

buffered PARASAL® TABLETS

the one dosage form for all your patients

BUFFERED PARASAL TABLETS automatically limit gastric hyperacidity, maintaining their buffering action for 2 to 3 hours. They do not produce "acid rebound" or interfere with normal gastrointestinal processes, and they are usually well tolerated even when other dosage forms are contraindicated.

For *your* patients with PAS-induced GI's, prescribe BUFFERED PARASAL TABLETS. Trial samples and literature will be gladly sent on request.



- EXTREMELY ECONOMICAL
- RAPIDLY ABSORBED
- SODIUM FREE
- SUGAR-FREE

Each capsule-shaped, uncoated tablet contains 0.5 Gm. Para-Aminosalicylic Acid in a special acid-buffering system.

THE Panray CORP.

340 CANAL ST., NEW YORK 13, N.Y.

When writing please mention *Diseases of the Chest*

Angiocardiography

Angiocardiography with Diodrast 70 per cent solution — whether by means of intravenous injection or by the technic of intracardiac catheterization — is now a well standardized and often immensely helpful procedure.

Diodrast

Concentrated Solution 70%

Ampuls of 20 cc. and 50 cc.

Specific instances in which it may be of unsurpassed diagnostic value are congenital lesions such as patent ductus arteriosus, tetralogy of Fallot, coarctation of the aorta, patent foramen ovale, dextrocardia, etc.; aneurysms, mediastinal lesions and chronic pericarditis.

Winthrop-Stearns INC.
New York 18, N. Y. Windsor, Ont.

Diodrast, trademark reg. U. S. & Canada, brand of iodopyracet

a new Picker technical publication

"HEART SIZE MEASUREMENTS IN CHILDREN"

In this new Picker publication Dr. Esguerra Gomez* extends the roentgenographic method of heart size prediction to include children. It is based on studies which have established a constant relationship between the heart diameters and the anthropometric index not only in adults, but in children as well. Nomograms and prediction tables, giving percentage deviations, eliminate the need for laborious calculations.

Dr. Gomez's work complements two earlier Picker technical texts – *Heart Size Measurements*, and *Roentgenology of the Heart* prepared by the Medical Department of the Equitable Life Assurance Society of the United States. Copies of any or all of them are available on request from the Picker X-Ray Corporation, as a complimentary service.

Dr. Gonzalo Esguerra Gomez
Clinica de Marly
Bogotá, Colombia, S. A.

Nomograms for area and transverse diameter of frontal heart silhouette, with interpretive key. Tables for theoretical transverse diameters for various heights and weights, with chart for determining percentage deviations from average.

A comprehensive discussion embracing techniques, interpretation of roentgenograms, and measurement formulae. Gives criteria for enlargement of cardiac chambers, and their significance in Heart Disease. With bibliography of Cardiac Roentgenology.

Picker x-ray

PICKER X-RAY CORPORATION
25 South Broadway, White Plains, N. Y.

DISEASES *of the* CHEST

VOLUME XXII AUGUST 1952 NUMBER 2

A Simple Method for the Determination of Vital Capacity—Time Relationships*

MAURICE S. SEGAL, M.D., F.C.C.P.,† J. AARON HERSCHFUS, M.D.†† and MAURICIO J. DULFANO, M.D.§
Boston, Massachusetts

The determination of vital capacity as an index of "pulmonary function" has been in use for more than a century. However, the vital capacity does not test function (dynamic), but only the difference between two static volumes; namely, between maximal inspiration and maximal expiration. Moreover, it has been observed that normal or high vital capacity values can sometimes be obtained in patients with obvious pulmonary disease. The value of a single determination has been compared to that of a single blood sugar determination.[1] The more definitive information obtained from a sugar tolerance study could comparably be obtained by determining the maximum breathing capacity.

The main shortcoming of the vital capacity as a pulmonary function test is its lack of time relationships. Several investigators[2-4] have included time relationships in determining the vital capacity on kymograph recordings and computed both inspiratory and expiratory velocity rates in terms of cubic centimeters per second. We have found such data of particular value in determining the relative efficacies of bronchodilator drugs in relieving bronchospasm.[4] Gross[5] also introduced a time relationship in his studies of the vital capacity in cardiac and pulmonary disease. He measured the time required for full maximal expiration,

*From the Department of Inhalational Therapy, Boston City Hospital, and the Department of Medicine, Tufts College Medical School.
†Clinical Professor of Medicine, Tufts College Medical School, and Director, Department of Inhalational Therapy, Boston City Hospital.
††Formerly Research Fellow in Medicine, Tufts College Medical School.
§Research Fellow in Medicine, Tufts College Medical School.

Copyright, 1952, by the American College of Chest Physicians

divided the vital capacity by that time and spoke of "expiratory velocity." Such a figure actually represented the average volume per second of expiration. Recently Gaensler[6] simplified the study of vital capacity - time relationships by adding a timing attachment to the Collins spirometer (Vitalometer). The timer can be set for 1, 2 or 3 seconds and the volume exhaled during one such period will be registered, as well as the total vital capacity. The vital capacity has to be repeated for each time interval that is set and the volume is read directly on the scale.

We are using a simple device, a Benedict-Roth recording spirometer* of nine liters capacity, whereby the volume exhaled during each second of a single vital capacity determination is recorded on the ventilograph paper and then measured. We prepared a transparent ruler* with parallel lines (Figure 1a); the distance between the lines corresponds to 1 second recording time on the

*Manufactured by Warren E. Collins, Inc., 555 Huntington Avenue, Boston, Massachusetts.

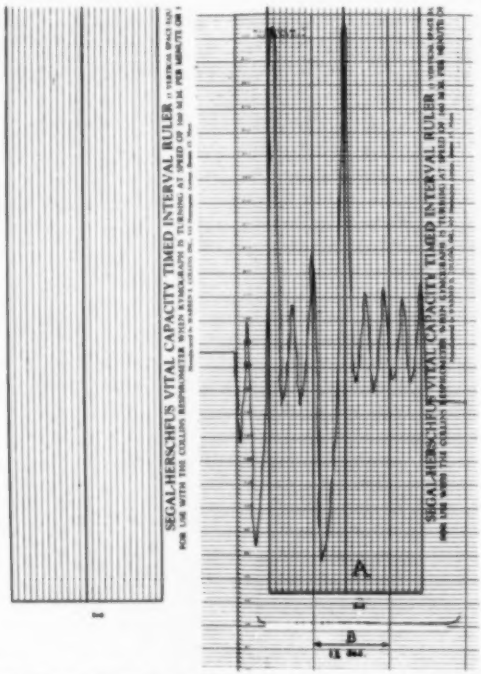


FIGURE 1: Transparency, which is divided by parallel lines into one-second intervals (a), is placed over the spirographic paper (b).

spirograph when turning at high speed (12 seconds between two heavy vertical lines on the ventilograph paper; Figure 1b). A vital capacity graph is recorded on the paper; the transparency is placed over the graph, and the volume during each second can be calculated. One millimeter vertical excursion of the recording needle equals 20.73 cubic centimeters. The ventilograph paper is lined with horizontal lines placed at two-millimeter intervals. Thus, to measure the *volume* for any one-second interval, the height of the tracing is measured in millimeters, multiplied by 20.73 and corrected for body temperature, barometric pressure and water vapor (B.T.P.S.). Both inspiratory and expiratory volume per second rates (cc./sec.) can be measured.

This method of time fractionation of the vital capacity has the advantages that the vital capacity is recorded graphically and that any desired time interval can be calculated from a single

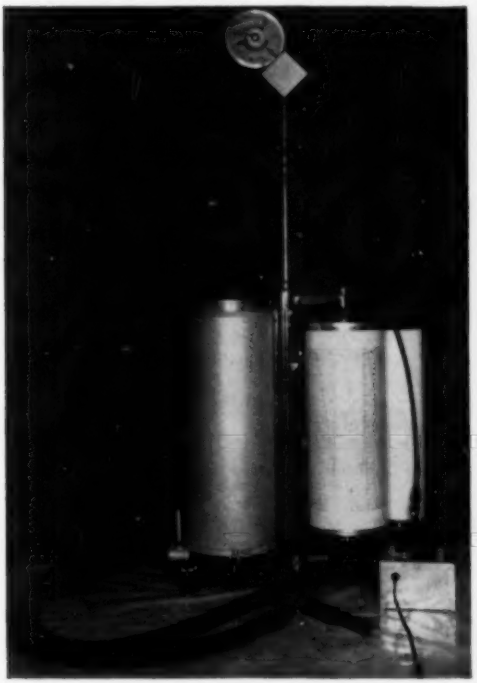


FIGURE 2: Recording Ventilograph* combined with the Solenoid Timing Device of the Vitalometer.*
(*Manufactured by Warren E. Collins, Inc., Boston, Mass.)

TABLE I

Analysis of Vital Capacity-Time Relationships Before and After Treatment with Bronchodilator Drugs in Two Patients, Comparing the Vitalometer Values with the Ruler-Ventilograph Values.

		First Second (cc.)		Total Vital Capacity (cc.)		1st Sec./Total V.C. x 100		Improvement (Per cent) — 1st Second		Improvement (Per cent) — Total V.C.	
		Vitalometer	Ruler-Graph	Vitalometer	Ruler-Graph	Vital.	R-G	Vital.	R-G	Vital.	R-G
(H.F.)	Before Treatment	500	525	1300	1281	38	41				
	After Treatment*	800	777	2300	2247	35	35	60	48	77	75
(A.McG.)	Before Treatment	1025	987	2300	2310	45	43				
	After Treatment**										
	1 Minute	1025	861	2300	2289	45	38	0	−1	0	−13
	5 Minutes	1900	1760	3400	3402	56	52	85	78	48	47
	10 Minutes	2000	1806	3750	3780	53	48	95	83	63	64

*Racemic Orthoxine, 50 mg. i.v.
**Neosuprel, 6 inhalations.

performance. The time intervals of the ruler are mathematically calculated and are not subject to fluctuations or mechanical sources of error. The observer should permit the kymograph to rotate for one or two seconds before starting the test in order to overcome initial inertia of the drum. In application of the ruler to the ventilograph there occasionally may be some difficulty in interpreting the exact point of intersection of the vertical line on the ruler with the graph.

Comparison with the Vitalometer.

The timing device was removed from the vitalometer, and attached to the Benedict-Roth recording spirometer. A recorded graph of the total vital capacity and scale readings of the first second expired volumes and total vital capacity were obtained simultaneously (Figure 2).

This permitted a comparison of our graphically recorded and measured method with the objective readings obtained from the mechanical solenoid timing device on the vitalometer. It should be noted that the readings on the scale of the vitalometer cannot be read accurately to within 50 cc.

With this combined apparatus, twelve determinations were made on normal subjects and 25 on asthmatic subjects. The total vital capacity as obtained directly from the spirographic tracing does not require the use of the ruler and is an accurate measurement. The total vital capacity readings obtained from the vitalometer were an average of 0.5 per cent smaller than those obtained simultaneously from the spirograph. The readings obtained from the first second of expiration with the vitalometer were an average of 11.3 per cent larger than those obtained with the ruler-graph technique. With the vitalometer the ratio of the first second to the total vital capacity was 59 per cent, and with the ruler-graph method, 55 per cent.

We also compared these two methods in studying the effects of bronchodilator treatment in two patients. The results appear in Table I. No striking differences were obtained between the two techniques. In general the vitalometer values tend to be somewhat larger.

SUMMARY

A simple ruler-graph device has been described for use with single determinations of the vital capacity on a recording drum apparatus. This permits the rapid determination of the volume of air ejected during each second of the vital capacity tracing on a graphic record. The same record also affords an opportunity

for analyzing the contour of the graph for evidence of trapping or change in mid-position.

These time relationships enhance the value of the simple vital capacity determination. The relative efficacies of bronchodilator preparations in relieving the bronchospasm of the patient with bronchial asthma or pulmonary emphysema can be determined readily with this technique.

This method was compared with the vitalometer. Both methods yield comparable values. The ruler-graph method, however, affords additional significant data, easily obtained by employing the apparatus which is generally in use in laboratories studying pulmonary function.

RESUMEN

Un método sencillo para determinar la capacidad se ha descrito, consistente en el uso de una regla gráfica en el tambor registrador del aparato. Esto permite la rápida determinación del volumen de aire expulsado durante cada segundo del trazo de la capacidad vital sobre un registro gráfico. El mismo registro también permite una oportunidad para analizar el contorno de la gráfica para descubrir la retención o el cambio en posición media.

Estas relaciones del tiempo aumentan el valor de las determinaciones de la capacidad vital simple. La eficacia relativa de las preparaciones broncodilatadoras para aliviar el broncoespasmo de los enfermos de asma o enfisema pulmonar puede ser determinada facilmente por este método.

Este procedimiento ha sido comparado con el vitalómetro. Ambos rinden valores comparables. El método de la regla-gráfica sin embargo, proporciona datos adicionales de significación que son obtenidos facilmente empleando el aparato que se usa generalmente en los laboratorios funcionales de pulmón.

RESUME

Les auteurs décrivent un appareil simple d'enregistrement par rouleau graphique pour déterminer la capacité vitale. Cet appareil permet de donner rapidement le volume de l'air rejeté à chaque seconde, lors du tracé de la capacité vitale. Ce même enregistrement offre la possibilité d'analyser l'aspect du tracé et de révéler la présence d'occlusions bronchiques ou d'altérations de caractère anormal.

Cette relation entre le tracé et la simple capacité vitale augmente la valeur de cette dernière. Elle permet de déterminer exactement avec cette technique l'efficacité relative des médications bronchodilatatrices, qui permettent de supprimer le bronchospasme chez les malades atteints d'asthme ou d'emphysème.

Cette technique est comparée au "vitalomètre." Les deux méthodes donnent des résultats comparables. Cependant, la méthode par rouleau graphique donne des renseignements supplémentaires, en employant l'appareil habituellement utilisé au laboratoire pour étudier la fonction pulmonaire.

REFERENCES

1 Wright, G. W.: Quoted in "Methods in Medical Research," Comroe, J. H., Jr., Editor, Yearbook Publishers, Chicago, 2:213, 1950.

2 Barach, A. L.: "Physiological Methods in Diagnosis and Treatment of Asthma and Emphysema," *Ann. Int. Med.*, 12:54, 1938.

3 Cournand, A., Richards, D. W., Jr. and Darling, R. C.: "Graphic Tracings of Respiration in Study of Pulmonary Disease," *Am. Rev. Tuberc.*, 40:487, 1939.

4 Segal, M. S.: "The Management of the Patient with Severe Bronchial Asthma," 1950, Charles C. Thomas, Springfield, Illinois.

5 Gross, D.: "Investigations Concerning Vital Capacity," *Am. Heart J.*, 25:335, 1943.

6 Gaensler, E.: "Analysis of the Ventilatory Defect by Timed Capacity Measurements," *Am. Rev. Tuberc.*, 64:256, 1951.

Venous Pressure as a Guide to Pneumoperitoneum Therapy in Pulmonary Emphysema*

GUSTAV J. BECK, M.D., F.C.C.P.,†
A. CHESMORE EASTLAKE, JR., M.D., F.C.C.P.† and
ALVAN L. BARACH, M.D., F.C.C.P.†
New York, New York

The striking improvement of symptoms in most patients with pulmonary emphysema treated with pneumoperitoneum has been correlated with changes in pulmonary function by Carter, Gaensler et al.[1,2] Although the circulatory changes in pulmonary emphysema have been the subject of intensive investigation,[3-7] there has been no report, so far, dealing with the hemodynamic effects of pneumoperitoneum in this disorder. The measurement of the venous pressure in the upper extremity in the course of pneumoperitoneum administration seemed to be one of the simplest methods to determine relative intrathoracic pressure changes. The results of these determinations are the subject of this preliminary report.

Methods

An unselected group of 10 patients, with marked pulmonary insufficiency secondary to far advanced pulmonary emphysema, was treated with pneumoperitoneum. In the course of their treatment, venous pressure determinations were performed during the administration of pneumoperitoneum. A calibrated Statham pressure transducer was used for continuous pressure registration which was recorded through a Sanborn Strain Gauge Amplifier on a Sanborn Visocardiette. The zero point of the transducer was chosen at a point 5 cm. below the costo-chondral junction of the second rib of the sternum. We were aware of the fallacy of this method for the determination of the level of the right auricle in patients with an increased anterior-posterior diameter of the chest due to pulmonary emphysema. Since we are, however, mostly interested in changes of venous pressure rather than absolute values, this method seemed adequate for our purpose. Peak pressures in inspiration and expiration and planimetrically calculated

*Presented at the Seventeenth Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 7-10, 1951.

†From the College of Physicians and Surgeons, Columbia University and the Presbyterian Hospital, New York.

mean pressures were averaged from the record during a control period of 15 minutes, following 30 minutes of complete bed rest, and after stabilization of the record following introduction of 300, 600, 900, 1200 and 1500 cc. of air in the abdomen. The symptom changes occurring during the administration of pneumoperitoneum were noted for correlation with the venous pressure changes. In two patients the experimental procedure was repeated and a total of 13 experiments will be reported.

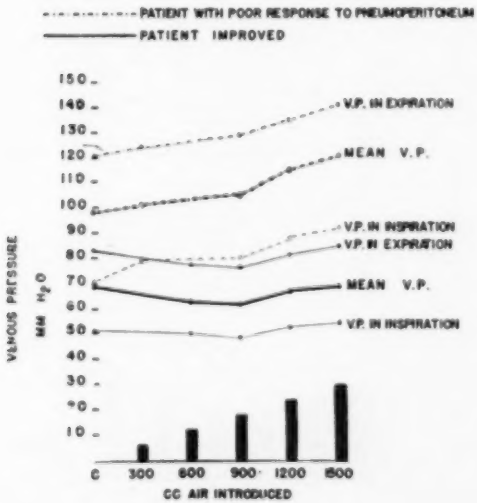
Results

Of 13 experiments there were six in which an average fall in mean venous pressure of 19 mm. of water (range −12 to −37 mm. water) occurred in the course of pneumoperitoneum. In three instances the change did not exceed ± 10 mm. of water (range −5 to +9 mm. water) (Figures 1, 2, 3, and 4).

In four experiments there was an average rise of mean venous pressure of 29 mm. of water (range plus 18 to plus 56 mm. water). In one patient with a significant drop in venous pressure at one time repetition of the experiment showed no change on another occasion. A second patient demonstrated no significant mean venous pressure change on one occasion and a significant rise during a subsequent determination.

FIGURE 1

VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEUM IN TWO PATIENTS WITH EMPHYSEMA



In eight experiments a rise occurred when 600 cc. of air or more was introduced into the abdomen. In only two cases did the rise exceed the control value of venous pressure (Figure 5).

All seven patients in whom nine experiments showed a drop or no change in venous pressure initially during pneumoperitoneum administration responded very well to this form of treatment clinically. There was markedly decreased dyspnea on rest and effort. In one of this group pneumoperitoneum had to be abandoned because of air in the scrotum. The one patient whose venous pressure did not change when pneumoperitoneum was first initiated had to be discontinued three months later, subsequent to the determination, during which he manifested a rise in venous pressure, because of increasing dyspnea following the introduction of air in the abdomen.

In the remaining three patients who manifested rises in venous pressure following pneumoperitoneum, this procedure had to be abandoned early because of severe respiratory embarrassment following the procedure. Two of these patients had slightly elevated venous pressures during the control period and in all three the

FIGURE 2

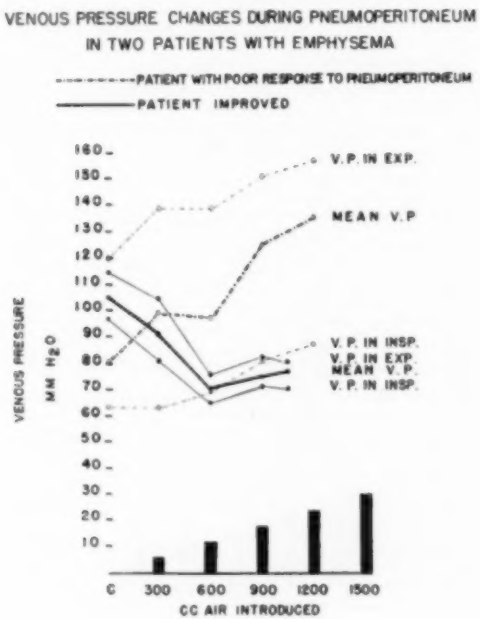


FIGURE 3

MEAN VENOUS PRESSURES DURING PNEUMOPERITONEUM
IN PATIENTS WITH PULMONARY EMPHYSEMA

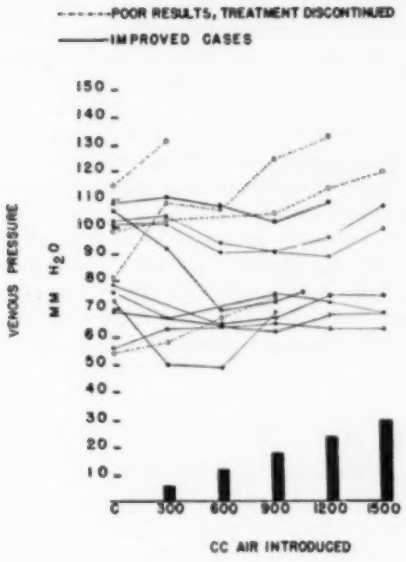
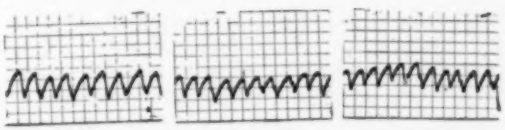


FIGURE 4

VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEM



INTRA-ABDOMINAL PRESSURE	+5 +7		+8 +10
INSPIRATORY V.P.	50.9 mm	48.4 mm	53.6 mm
Expiratory V.P.	83.6 mm	76.1 mm	81.5 mm
VENOUS PULSE PRESSURE	32.7 mm	27.7 mm	27.9 mm
MEAN V.P.	68.9 mm	62.1 mm	67.6 mm

venous pressure rose to 120 mm. of water or more following the introduction of air into the abdomen.

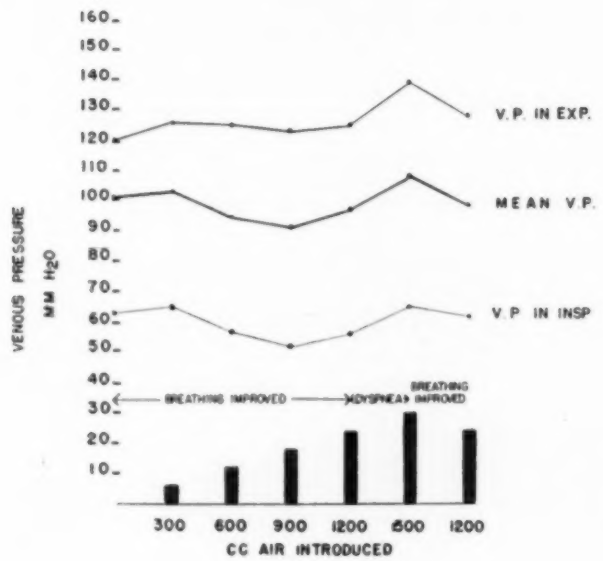
Increased dyspnea also occurred in the group which responded well to treatment when in seven of the nine experiments a venous pressure rise occurred with increased air in the abdomen, after the venous pressure had reached its optimum low level. The symptoms could be alleviated by removal of some of the excess air from the abdomen at the end of the procedure.

Discussion

By introducing 100 cc. of air into the peritoneum of normal animals, Overholt[8] has shown that the intra-abdominal pressure can be raised from subatmospheric levels to above atmosphere. Pressure tracings revealed inspiratory and expiratory intra-abdominal pressure changes which paralleled the respective intrapleural pressures. The presence of intrathoracic hypertension in emphysema was demonstrated experimentally by Kauntz and his group[9] when they obtained an increase in intrapleural pressure

FIGURE 5

VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEUM IN A PATIENT WITH PULMONARY EMPHYSEMA



and venous pressure in dogs with partial obstruction of expiration over a period of weeks, in which partial enlargement of the lungs occurred. Duomarco and his collaborators[10] demonstrated the existence of abdominal hypertension in subjects with intrathoracic hypertension, thus demonstrating a close relationship between intrapleural and intra-abdominal pressure in patients with uncomplicated emphysema.

A discrepancy between changes in intra-abdominal and intrathoracic pressure in patients with pulmonary emphysema was observed by Alexander.[11] A belt was applied to the lower part of the abdomen, thus increasing the intra-abdominal pressure in emphysematous patients. The elevation of the diaphragm caused increased excursion of the leaves and restoration of the previously elevated intrapleural pressure to normal subatmospheric levels. This change was accompanied by a drop in venous pressure. Symptomatic relief occurred in these patients.

In some cases of emphysema, pneumoperitoneum or increased abdominal pressure exerted by an abdominal belt produces a lowering of the venous pressure as a result of decreasing intrapleural pressure. A more advantageous mechanism for ventilating the lungs is produced in that the ventilating cycle is accomplished by contraction of the uplifted diaphragm rather than by contraction of abdominal muscles and lower intercostals to create increased intra-abdominal pressure. Ventilation then occurs with an intrapleural pressure swing below the atmosphere rather than partly above and partly below. In these cases contraction of the diaphragm is followed by expansion of the lung.

In those cases in which the venous pressure is elevated, the uplifted diaphragm does not contract and elevate, either because of atrophic changes in the diaphragm or inadequate elasticity in the lungs. The pressure is, therefore, transmitted to the lungs and initiates effects similar to pressure breathing. In a paper by Barach, Martin and Eckman[12] in which venous pressure changes were observed as a result of pressure breathing, an increase in venous pressure of 40 per cent of the applied pressure was found in normal subjects, but in patients with congestive heart failure, in whom there was decreased pulmonary elasticity, the rise in venous pressure was in the neighborhood of 70 per cent of the applied pressures. In addition, marked prolongation of circulation time took place in the subjects with heart disease. It would appear likely that patients in whom a rise in venous pressure, instead of a fall, took place after pneumoperitoneum or after an excess of air was introduced, would endure the same physiologic effects as pressure breathing, namely, a blockade of blood into the right heart with a slowed circulation time and decreased cardiac output.

Under these circumstances, a stagnant type of anoxia would contribute to dyspnea. The factor of the function of the right heart in delivering blood to the lungs at an increased intrapulmonary pressure must also be considered. In those instances in which right ventricular insufficiency is present, either overt or latent, the increased venous pressure may develop in part as a result of inability to empty the right ventricle under conditions of increased intrapulmonary pressure.

This is in agreement with the observations of Gillanders,[7] who found that no rise in venous pressure was obtained when pressure was applied to the abdomen of normal or uncomplicated emphysematous subjects; whereas, in the presence of right side heart failure in patients with emphysema, this maneuver led to a marked rise in venous pressure.

Training in diaphragmatic breathing has recently been emphasized as a factor of considerable value in the treatment of patients with pulmonary emphysema.[13,14] Conceivably, the decreased tone of the diapnragm in those cases in which diaphragmatic breathing has not taken place, may account for the failure of the development of a negative pressure or an increased negative pressure within the lungs after pneumoperitoneum. If increased diaphragmatic training were to take place, patients of this kind may, on subsequent trials of pneumoperitoneum be found to benefit by the procedure as a result of more efficient contraction of the elevated diaphragm.

SUMMARY

1) Venous pressures were determined during the administration of pneumoperitoneum in 10 patients with pulmonary emphysema.

2) Six patients showed a consistent drop or no change in venous pressure. All derived excellent benefits from pneumoperitoneum.

3) In three patients in whom a rise in venous pressure was recorded, pneumoperitoneum had to be discontinued because of respiratory embarrassment following it.

4) One patient who on one occasion had no change in venous pressure tolerated pneumoperitoneum well for three months. Subsequently, dyspnea became so incapacitating following the procedure that it had to be abandoned. A rise in venous pressure was noted during the last pneumoperitoneum.

5) An increase in diaphragmatic excursion is felt to be responsible for the lowering of venous pressure in the successful cases of pneumoperitoneum therapy.

6) Direct transmission of the intra-abdominal pressure increase to the intrapleural space through a fixed atrophic diaphragm, in the presence of markedly reduced pulmonary elasticity, blocks the

flow of blood into the right heart with resultant increased venous pressure, decreased right cardiac output and stagnant anoxia. This accounts for the respiratory embarrassment in these patients following pneumoperitoneum.

7) Changes in venous pressures during pneumoperitoneum seem to be a good prognostic index for emphysematous patients in whom this form of treatment is contemplated.

RESUMEN

1) Las presiones venosas fueron determinadas durante la administración de neumoperitoneo, en 10 pacientes con enfisema pulmonar.

2) Seis enfermos mostraron una baja continua o ningún cambio en la presión venosa. Todos obtuvieron excelentes beneficios del neumoperitoneo.

3) En tres de ellos en los cuales se notó un aumento en la presión venosa, el neumoperitoneo tuvo que ser suspendido debido al impedimento respiratorio que le siguió.

4) Un paciente que en una ocasión no sufrió cambio en la presión venosa, toleró el neumoperitoneo bien por tres meses. Mas tarde, la disnea vino a ser tan incapacitante al seguir el procedimiento, que éste tuvo que ser abandonado. Un aumento en la presión venosa fué notado durante el último neumoperitoneo.

5) Se cree que un aumento en la excursión del diafragma es el responsable de la baja de la presión en los casos con éxito de terapéutica del neumoperitoneo.

6) La transmisión directa de la presión intra-abdominal aumenta hacia el espacio intrapleural a traves de un diafragma atrófico fijo, en la presencia de una reducción marcada de la elasticidad pulmonar, perjudica el paso de la sangre al corazón derecho con el aumento resultante de presión venosa, decrecimiento del rendimiento cardiaco y la anoxia por éxtasis. Esto produce el impedimento respiratorio que siguió al neumo-peritoneo en esos pacientes.

7) Parece ser que los cambios en la presión venosa durante el neumoperitoneo son un buen índice de pronóstico para los pacientes enfisematosos en los cuales esta forma de tratamiento es usada.

RESUME

1) On a mesuré la pression veineuse au cours de l'exécution d'un pneumopéritoine chez 10 malades atteints d'emphysème pulmonaire.

2) Chez six malades, on constata soit une chute nette, soit une absence de transformation de la pression veineuse. Tous tirèrent un très grand bénéfice de l'action du pneumopéritoine.

3) Chez trois d'entre eux, on constata une augmentation de la pression veineuse. Il fallut alors cesser le pneumopéritoine, car il était suivi de troubles respiratoires.

4) Chez l'un des malades, qui n'avait pas eu de modification de la pression veineuse, le pneumopéritoine fut parfaitement toléré pendant trois mois. Ultérieurement la dyspnée consécutive au pneumopéritoine était telle qu'on dut l'abandonner. Au cours de la dernière insufflation du pneumopéritoine, on constata une augmentation de la pression veinuse.

5) Les auteurs pensent que l'augmentation de l'élévation diaphragmatique est la cause de l'abaissement de la pression veineuse dans les cas où le pneumopéritoine a donné de bons résultats.

6) La transmission de l'augmentation de la pression abdominale se propage à l'espace intra-pleural à travers le diaphragme s'il est fixé et atrophique, et la réduction notable de l'élasticité pulmonaire ploque le flux sanguin dans le coeur droit. Il en résulte une augmentation de la pression veineuse, une diminution de débit du coeur droit et une anoxie de stase. Ceci rend compte des troubles respiratoires qui sont, chez ces malades, consécutifs au pneumopéritoine.

7) Les modifications de la pression veineuse au cours du pneumopéritoine semblent être un élément favorable de pronostic chez les malades emphysémateux qui sont soumis à ce traitement.

REFERENCES

1 Carter, M. G., Gaensler, A. E. and Kyllonen, A.: "Pneumoperitoneum in the Treatment of Pulmonary Emphyesma," *New England J. Med.*, 243:549, 1950.
2 Gaensler, A. E. and Carter, M. G.: "Ventilation Measurements in Pulmonary Emphysema Treated with Pneumoperitoneum," *J. Lab. Clin. Med.*, 35:945, 1950.
3 Blumgart, H. L. and Weiss, S.: "Clinical Observations on the Velocity of Blood Flow in Auricular Fibrillation and in Emphysema," *Trans. Assoc. Am. Phys.*, 41:94, 1926.
4 Kauntz, W. B., Pearson, E. F. and Koenig, K. F.: "Observations on Intrapleural Pressure and Its Influence on the Relative Circulation Rate in Emphysema," *J. Clin. Inv.*, 11:1281, 1932.
5 McMichael, J.: "Circulatory Failure," *Schweiz, Med. Wchnschr.*, 76: 851, 1946.
6 Hickman, J. B. and Cargill, W. H.: "Effect of Exercise on Cardiac Output and Pulmonary Arterial Pressure in Normal Persons and in Patients with Cardiovascular Disease and Pulmonary Emphysema," *J. Clin. Inv.*, 27:10, 1948.
7 Gillanders, A. D.: "Circulatory Dynamics in Emphysema," *Quart. J. Med.*, 18:263, 1949.
8 Overholt, R. H.: "Intraperitoneal Pressure," *Arch. Surg.*, 22:691, 1931.
9 Kauntz, W. B., Alexander, H. L. and Dowell, D. J.: "Emphysema Simulating Cardiac Decompensation," *J. Am. Med. Assn.*, 93:1369, 1929.
10 Duomarco, J., Rimini, R. and Recarte, Y. P.: "Intra-abdominal Pressure in Emphysema and in Congestive Cardiac Insufficiency with Reference to the Problem of Thoracic Hypertension," *Arch. urug. de Med. Cir. y especialid.*, 25:244, 1944.
11 Alexander, H. L.: "The Treatment of Emphysema," *Internat'l. Cl.*, 4: 211, 1936.

12 Barach, A. L., Martin, J. and Eckman, M.: "Positive Pressure Respiration and Its Application to the Treatment of Acute Pulmonary Edema," *Ann. Int. Med.*, 12:754, 1938.
13 Barach, A. L.: "Physiologic Methods in the Diagnosis and Treatment of Asthma and Emphysema," *Ann. Int. Med.*, 12:454, 1938.
14 Barach, A. L.: "Physiologic Treatment of Pulmonary Emphysema," *Am. Prac.*, 2:771, 1948.

Discussion

LEON H. HIRSH, M.D., F.C.C.P.
Milwaukee, Wisconsin

I have been using pneumoperitoneum for this purpose in my office and in the emphysema clinic at Mount Sinai Hospital in Milwaukee for the past two years. Venous pressure measurements have been made with a water manometer before and periodically during the pneumoperitoneum regimen. Our experience parallels that he presented.

With reference to the volume of the pneumoperitoneum inflations we have arrived at the same conclusions emperically as the authors. That is to say, patients derive greater benefit from small inflations than large.

Of 17 patients with chronic so-called hypertrophic pulmonary emphysema 11 received pneumoperitoneum. One suffered acute appendicitis after six months of pneumoperitoneum. Inflations were necessarily discontinued during surgery and convalescence. They were reestablished four weeks after appendectomy. In the interim, there was recurrence of symptoms of emphysema.

All 11 have enjoyed a favorable response to pneumoperitoneum. One has reported slight relief and 10 enjoy satisfactory relief. To offer an idea of satisfactory relief, may I cite the following cases: One became totally disabled with emphysema and was obliged to sell his welding shop and his home. He moved to a trailer and was unable to walk its length without dyspnea and cyanosis. Today he is building a house. A few weeks ago he changed the motor in his car. Another with emphysema spent an entire year before pneumoperitoneum confined to his home with oxygen continuously available. Since pneumoperitoneum he has dispensed with the oxygen tank. At first he required transportation to the clinic in an automobile, but now he comes in a street car.

One may readily understand the rationale of therapeutic pneumoperitoneum when considering the anatomic and functional changes characteristic of pulmonary emphysema: (1) There is destruction of the perialveolar and peribronchial elastic fibers; (1) there is rupture of large numbers of alveoli; (3) the intra-

pleural negative pressure becomes less negative or even atmospheric; (4) there is a loss of the contripital contractility of the lung; (5) the diaphragm occupies a constant low inspiratory position. It is well known that the diaphragm is responsible for from 37 to 47 per cent of the ventilatory function of the lungs; (6) the return of the venous blood from the periphery to the right side of the heart is decreased; and (7) the blood flow from the right side of the heart to the lung is diminished.

Dyspnea and cyanosis result from the loss of functionally competent alveoli, dislocation of the diaphragm downward, and interference with the normal circulation of the blood from the periphery to the heart and from the heart to the lung.

Empirical observations prove that artificial pneumoperitoneum corrects the functional derangements responsible for the clinical manifestations of pulmonary emphysema. This may be partial or complete. Injection of moderate amounts of air into the abdominal cavity causes a rise of the diaphragm which is brought about by the following factors: (1) Neutralization of the sub-diaphragmatic negative pressure. (2) Support of the pulling effect of the intrapleural negative pressure. (3) The diaphragm is displaced upward by the positive intra-abdominal pressure.

When the diaphragm approaches or is restored to its normal position the intrapleural pressure becomes more negative than prior to treatment. The diaphragm regains its piston-like motion during the two respiratory phases. The venous blood flow from the periphery to the heart is facilitated together with an accelerated flow of blood from the right side of the heart to the lung.

These changes are easily demonstrated clinically and herein lies the rationale for the use of therapeutic pneumoperitoneum in hypertrophic pulmonary emphysema.

Physiologic Factors in the Use of the Body Respirator for Impaired Respiratory Function*

JAMES L. WHITTENBERGER, M.D. and
JAMES V. MALONEY, JR., M.D.
Boston, Massachusetts

It is of interest that the history of body-enclosing chambers for the treatment of respiratory diseases goes back to at least 1885, when H. F. Williams, an American physician, aided by an engineer friend, used this principle in administering aerosol therapy to patients with pulmonary infections.[1] A year later Williams reported that cyclic changes in pressure around the body could be used to produce passive respiration and to assist breathing in diseased conditions.[2] This experience was forgotten except in a few physiology laboratories where body plethysmographs were used to study respiration and produce ventilation in experimental animals.[3] The idea was rediscovered in 1927 by Drinker and Shaw, and following their successful application of a respirator in treating poliomyelitis patients[4] its use has spread to the treatment of other types of respiratory failure.

Before considering physiologic aspects of the respirator, it is pertinent to review certain characteristics of the normal mechanics of breathing, gas exchange, and pulmonary circulation, since all of these components of external respiration are inseparably related to the interchange of carbon dioxide and oxygen between the body and the atmosphere. Likewise, all three components of external respiration are involved to various degrees in pulmonary diseases and are affected adversely or beneficially by the methods of therapy. Since the object of this report is consideration of the use of the respirator, most attention will be directed to the mechanics of breathing, but it must be kept in mind that mechanical ventilation of the lungs has little purpose except the regulation of alveolar gas concentrations, and the pressure changes in the thorax associated with ventilation, whether due to muscular activity or to a mechanical device, have inevitable effects on the circulation of blood through the thorax.

Lung ventilation in the normal organism is so regulated that the alveolar partial pressure of carbon dioxide seldom varies

---

*From the Department of Physiology, Harvard School of Public Health, Boston, Massachusetts.

Presented at the 17th Annual Meeting of the American College of Chest Physicians, Atlantic City, New Jersey, June 8, 1951.

more than 3 mm. from the mean value of 38 mm. Hg., at sea level. At higher altitudes the alveolar pressure of carbon dioxide is stabilized at a lower level. When the carbon dioxide partial pressure varies beyond these narrow limits, alkalosis or acidosis occurs. If the abnormal value continues for many hours or days, adjustments in the bicarbonate levels of the body occur so that the pH is returned toward normal. After a few days of the abnormal carbon dioxide level, the respiratory center undergoes a change in sensitivity so that it tends to maintain ventilation at the new level.[5] Of most practical importance is this sort of change in the emphysema patient whose chronic underventilation leads to accumulation of high concentrations of carbon dioxide in the body.

The fine control of carbon dioxide concentration in the body is not imitated in the case of oxygen. Because of the shape of the oxygen-hemoglobin dissociation curve an almost normal load of oxygen can be picked up by the blood passing through the lungs, although lung ventilation, and hence oxygen concentration, may be considerably less than normal. High concentrations of inspired oxygen, up to 60 per cent at one atmosphere, also have little effect on the body (other than to overcome an increased diffusion barrier). In fact, lung ventilation is not essential for supplying oxygen to the body, if the organism is under basal conditions, pure oxygen is administered, and the nitrogen is first flushed out of the system. This phenomenon has come to be known as "diffusion respiration."[6] Although adequate for oxygenation, the method is not practical because in the absence of pulmonary ventilation carbon dioxide concentration soon reaches a narcotic level.

It is apparent, then, that mechanical treatment of respiratory failure is primarily to regulate carbon dioxide exchange, and that this exchange is ordinarily controlled within very narrow limits by the respiratory regulatory mechanisms. If carbon dioxide exchange is properly regulated and the patient continues to show signs of hypoxia, the concentration of inspired oxygen must be raised.

The normal active breathing movements are, like most integrated activities of the body, exceedingly complex in detail but simple enough in broad outlines. When all respiratory muscles are relaxed at the end of an expiration, the lungs have a certain volume, known usually as the functional residual volume, and in terms of the mechanics of respiration as the *relaxation volume*. This volume represents an important base line and is the volume at which the tendency of the lungs to contract is exactly balanced by the tendency of the thorax to expand. These opposing forces

result in the static negative interpleural pressure. The major forces involved are due to elastic properties of lungs and chest, plus the action of gravity on abdominal contents and diaphragm. It is worth emphasizing that the chest has a natural tendency to expand to a volume somewhere between 50 and 70 per cent of the normal vital capacity scale.[7] This degree of expansion is usually reached in advanced emphysema when the counterforce of an elastic lung is largely lost.

In normal breathing the thorax is actively expanded by muscular action, thereby further reducing interpleural pressure. The associated reduction of pressure in the alveolar spaces results in a pressure gradient in the tracheobronchial tree, and air flows into the lungs until alveolar pressure approaches atmospheric pressure. The transpulmonic pressure difference at this point is greater than before since the lung is more stretched; in other words, the interpleural pressure is more negative. Usually, muscular relaxation starts before inspiratory air flow stops, interpleural pressure becomes less negative, and expiration quickly proceeds, assisted greatly by the elastic recoil of the lungs. In quiet unobstructed breathing, expiration ends when the opposing elastic and gravitational forces come into equilibrium.

Ventilatory failure may result from a variety of causes, including muscular paralysis, fatigue, skeletal deformities, tracheobronchial obstruction, and increased resistance to lung tissue deformation. Often there is a combination of causes. The aim of therapy is to replace or supplement the inadequate motor mechanism; the method is basically simple—the rhythmic creation of a pressure difference between the alveolar spaces and the outside of the thorax, sufficient to distend the lungs by the desired amount. The pressure difference upon which lung inflation depends may be created either by increasing pulmonary pressure, as in ordinary pressure breathing, or by reducing pressure outside the body, as in the respirator. The only significant difference between the two methods is that the gas density changes in the first method account for approximately 10 per cent more tidal exchange in the case of conventional pressure breathing.[8] The importance of recognizing the essential similarity of the two methods is that the large body of evidence on the physiologic effects of pressure breathing can be applied to the therapeutic use of the body respirator. The circulatory effects of pressure breathing, as summarized by Barach, Fenn, Ferris, and Schmidt[9] are primarily the reduction of venous return to the right heart (usually temporary), rise in systemic venous pressure, increased systemic capillary filtration, reduction in blood volume, systemic arteriolar constriction, and elevation of cerebrospinal fluid pressure. Barach, Martin,

and Eckman recognized the importance of these factors in the treatment of pulmonary edema.[10] In addition, the increase in functional residual volume and widening of the bronchial channels associated with certain types of pressure breathing have led to beneficial results in the treatment of asthma.[10,11]

Comparative Advantages of Different Methods of Pressure Breathing. Both methods of creating a pressure difference have been used extensively in the treatment of respiratory paralysis of central or peripheral origin. In general the body respirator has been adopted in treating prolonged respiratory paralysis, as in poliomyelitis and other neuromuscular defects of the respiratory motor apparatus. The most significant advantages of the body respirator are its safety and the fact that the head is free of mechanical appliances. Thus eating, drinking, talking, and aspiration procedures can be done without interruption of artificial respiration. It is difficult to gain these advantages with any form of mask, hood, or dome used independently of a respirator. On the other hand the body respirator handicaps nursing and other therapeutic procedures, requires at least a semirecumbent position, has a set pattern of cycling pressures, and has a limited range of cycling rates.

For short term respiratory paralysis or as an accessory to the body respirator, various means of applying pressure at the face are widely used. The intermittent positive pressure and alternating positive and negative pressure resuscitators apply pressure by means of a face mask; they adequately breathe the patient when properly used but are not suitable for round-the-clock dependability. Other devices have been used to supplement the pressure differential created by the body respirator, or to substitute for the respirator when free access to the body is needed. The former type includes bellows assemblies operating in phase with the respirator, supplying air or high oxygen mixtures to the airway through tracheotomy tube adapters, face masks, hoods, or plastic domes. The principles are the same for all types of pressure breathing.

Effects on Ventilation

A. *Tidal exchange when air channels are unobstructed.* The patterns of pressure change in most respirators are such that air flow proceeds almost to completion during the inspiratory phase of the cycle. Under these circumstances tidal exchange is maximal for a given pressure setting, since alveolar pressure approximates atmospheric when the respirator is at its negative peak. However, the effectiveness of the transpulmonic pressure difference is modified by the visco-elastic properties of lungs and

thorax, and by reflex resistance if the muscles are not paralyzed. Unfortunately, the variability of these properties in patients with emphysema, poliomyelitis, and other diseases makes it impossible to predict with accuracy what tidal ventilation will result from a given pressure setting. In the absence of ventilation measurements or blood gas data, physicians have had to rely upon the subjective reactions of the patient or the empiricism of experience. It is presumptuous to expect clinical judgment to be a perfect substitute for the body's complex neurochemical regulation of ventilation.

B. *Tidal exchange in the presence of airway obstruction.* With increased tracheobronchial resistance the flow of air into the lungs is slowed and the alveolar spaces do not have time to fill to atmospheric pressure before the peak negative respirator pressure is passed. The transpulmonic pressure difference is less than the cycling pressure range of the respirator, and the ventilation is less than would be expected from the respirator gage setting. The remedy is to alleviate the obstruction if possible, or to increase the pressure range until adequate ventilation is obtained.

As in asthmatic patients, achieving adequate ventilation in obstructed patients is not without cost. The respirator produces active inspiration, as in normal breathing, and expiration must occur by elastic recoil of the lungs, supplemented by active expiratory effort if the patient is able. In the presence of obstruction, there may not be time for expiration of a normal tidal volume, so that inspiration starts from a higher point on the lung volume scale. This process of increasing functional residual volume will continue until there is sufficient elastic recoil in the overstretched lungs to expel in the time available an amount equal to the inspired volume. If a further analogy to asthma is permissible, one would expect this situation to result eventually in loss of lung elasticity. The increase in functional residual volume can be prevented by augmenting the positive pressure phase inside the respirator, to assist expiration.

C. *Effects of the respirator on pulmonary secretions and transudates.* It is not appropriate to discuss here the theory of treating lung edema by pressure breathing, except to say that there is sound evidence for desirable effects on the mechanics of breathing, on gas exchange, and on the circulation, when suitable applications of pressure are made. As a means of giving pressure breathing to treat pulmonary edema, the respirator excels, in that a wide variety of pressure combinations is possible. It has the disadvantages of not responding to the patient's breathing desires, if present, and of requiring the recumbent position. The first disadvantage is eliminated in a respirator to be described in a

later paragraph; the second handicap would be technically easy to eliminate.

Some individuals believe that the body respirator may cause aspiration of secretions into bronchi and alveolar spaces, resulting in alveolar edema and atelectasis, brought about in some way by the negative pressure. Some of these statements arise from a misconception about the nature of respirator operation, namely, failure to realize that the respirator has the same effects on tracheo-bronchial diameter and lung volume as normal breathing. Normal breathing with the same tidal volume should have the same effects on movement of secretions. If pressure breathing is useful in treating atelectasis, the respirator is likewise useful, although higher than average pressure gradients may have to be used.

Effects of the respirator on circulatory function. That the respirator has the same effects on the circulation as other forms of pressure breathing has been experimentally demonstrated.[8] Elevation of airway pressure above the ambient pressure of the rest of the body impedes venous return and cardiac output, at least during the period of elevated airway pressure. This effect is of little or no consequence if the high airway pressure phase is short enough or if compensatory circulatory mechanisms produce a rise in systemic venous pressure. Failure of the compensatory response occurs in the presence of vasomotor paralysis or shock accompanied by low blood volume. In these circumstances the body respirator has been found to depress the circulation in man and in experimental animals.[12] When expiratory positive pressure in the respirator is used to reduce the required degree of negative pressure, giving a mean pressure near atmospheric, the circulation is improved.

Another mechanism by which circulatory impairment can occur is that of gastric distention by air pressure. When lung ventilation is brought about by anesthesia bag compression, it is not rare to find that air has been forced into the stomach as well as the lungs: this complication is likely to occur when airway resistance is increased. Increase in gastric air has been observed in respirator patients, brought about by the same mechanism.

A Patient-Cycled Body Respirator. In addition to its use in treating respiratory paralysis, the body respirator has given excellent results, on a limited scale, in the treatment of cardiac pulmonary edema[13] and the impaired respiratory function of emphysema.[14] These conditions have been treated on a large scale by apparatuses applying pressure to the face.[10,15] The latter have the advantage of cycling in response to the patient's breathing desires, but are difficult to maintain in use for long. It has been

considered desirable to combine the advantages of the two methods, by having a body respirator cycle in response to the patient's desires, and at the same time having the face free. Such a respirator would have potential usefulness not only in treating patients with asthma, emphysema, and pulmonary edema, but in treating victims of bulbar poliomyelitis, in whom breathing impulses are present but erratic, conflicting with regular pulsations of the conventional respirator.

In response to this need the J. H. Emerson Company has developed a respirator attachment which permits a patient to cycle the body respirator at almost any desired rate with very little effort. Any type of body-enclosing chamber may be used, the pressure fluctuations being obtained by connecting the chamber to a vacuum-blower.* The effectiveness of the suction is determined by a solenoid-operated valve which in turn is controlled by the cycling mechanism attached to the patient. When the valve is closed, air is evacuated from the chamber, resulting in a steady fall of pressure until the valve opens. When the valve is open, air rushes back into the chamber, raising the pressure to atmospheric. The cycling mechanism consists essentially of a pressure-sensitive switch, which is opened or closed by the breath-

*Spencer Turbo-Compressor, capacity 50 cu. ft. per minute.

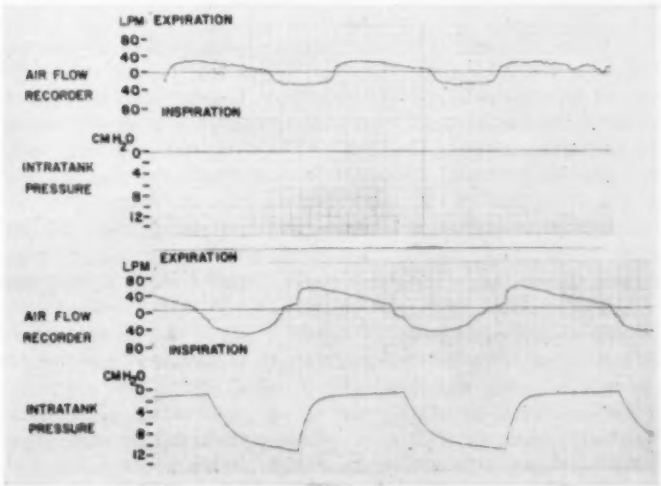


FIGURE 1: Time relationships of respiratory air flow rate and intratank pressure. Upper tracing, spontaneous breathing, tank not operating; lower tracing shows increase in ventilation when tank is operating.

ing movements. A pressure change of only a fraction of a millimeter of water is sufficient to trip the switch. This pressure change is readily obtainable from an ordinary pneumograph cuff around the chest or abdomen, or it may be obtained by holding the open end of the transmitting tube in the air stream at the mouth or one of the nares. The switch is set so that the solenoid-operated valve is open at the rest position. With this setting slight inspiratory effort causes closure of the solenoid-operated valve and the intratank pressure starts to fall, thereby assisting inspiration. When lung inflation is completed and air flow stops, the solenoid-controlled valve automatically opens and expiration occurs essentially at atmospheric pressure.

The time relationships of respiratory air flow rate and intratank pressure in a normal subject are shown in Figure 1. The top tracing indicates that in quiet spontaneous breathing there is a sustained inspiratory flow of 25 to 30 liters per minute. The lower tracing was made with the mechanism operating; it is evident that tank pressure starts to fall when inspiratory flow rate reaches a level between 10 and 20 liters per minute, at a time when only a small fraction of a normal tidal volume has been inspired. Inspiration then continues at higher than normal flow rates, producing hyperventilation similar to that seen in the first few moments of any type of pressure breathing.

Subjectively, the application of pressure in this way is comfortable. There is not only freedom from the discomfort of a tightly-fitted mask, but there is no added dead space effect to be overcome by increased tidal volume. The patient-cycled respirator has been used on two patients convalescing from poliomyelitis and on one patient in status asthmaticus. Data on these and other patients will be reported subsequently.

SUMMARY

In normal breathing the respiratory muscles change the configuration of the thorax in such a way that air inflates the lungs on each inspiration and is expelled by elastic recoil of the lungs on each expiration. These movements not only effect gas exchange but affect the circulation of blood through the thorax. If the respiratory muscles are for any reason inadequate, their efforts must be assisted or replaced by mechanical means. The pulmonary effects of normal breathing can be very nearly simulated by intermittent elevation of airway pressure (obtained by raising pulmonic pressure with the body at atmospheric pressure, or by lowering pressure around the body with the lungs remaining at atmospheric pressure). The effects of intermittent positive pressure breathing on the circulation are very different from those

of normal breathing; the impeding effect on cardiac output is beneficial in some conditions (pulmonary edema) and detrimental in others (shock). The advantages of alternative methods of pressure breathing are presented. A device which permits the patient to cycle a body respirator is described.

RESUMEN

En la respiración normal los músculos respiratorios cambian la configuración del tórax de tal manera que el aire infla los pulmones en cada inspiración y es expelido por retracción elástica de los pulmones en cada expiración. Estos movimientos no solo afectan el intercambio de gas sino también la circulación de la sangre a traves del tórax. Si los músculos respiratorios son inadecuados por cualquier motivo sus esfuerzos deben ser ayudados o reemplazados por medio mecánicos. Los efectos pulmonares de la respiración normal pueden ser casi simulados por la elevación intermitente en la presión de las vías aereas (obtenida por la elevación de la presión pulmonar con el cuerpo a presión atmosférica, o por descenso de la presión alrededor del cuerpo con los pulmones permaneciendo a presión atmosférica). Los efectos de la respiración a presión intermitente positiva sobre la circulación son muy diferentes de los de respiración normal; el impedimento del rendimiento cardiaco es benéfico en ciertas condiciones (edema pulmonar) y deprimente en otras (shock). Las ventajas de los métodos alternantes de la respiración a presión son presentadas. Se describe una innovación que permite al paciente arreglar los ciclos de un respirador corporal.

RESUME

Dans la respiration normale les muscles respiratoires modifient la configuration thoracique. Ainsi l'air gonfle les poumons à chaque inspiration et est chassé par la rétraction élastique des poumons à chaque expiration. Ces mouvements ne concernent pas seulement les échanges gazeux, mais ont également leur incidence sur la circulation du sang à travers le thorax. Si les muscles respiratoires sont incapables, pour une raison ou une autre, de remplir leur mission, il faut les aider ou les remplacer à l'aide de moyens mécaniques. L'action de la respiration normale sur le poumon peut être artificiellement obtenus de façon très proche de la normale, par l'élévation intermittente de la pression de l'air. Les effets sur la circulation de la respiration en pression positive intermittent sont très différents de ceux de la respiration normale. L'action sur l'insuffisance cardiaque, très heureuse dans certains cas (oèdème pulmonaire) est fâcheuse dans d'autres (choc). Les auteurs montrent les avantages de la respiration en pression alternative.

REFERENCES

1 Williams, H. F.: "Antiseptic Treatment of Pulmonary Disease by Means of Pneumatic Differentiation," *M. Record*, 27:57, 1885.
2 Williams, H. F.: "The Progress of Pneumatic Differentiation," *J.A.M.A.*, 7:169, 1886.
3 Chillingworth, F. P. and Hopkins, R.: "Physiologic Changes Produced by Variations in Lung Distention. A Body Plethysmograph and Its Application to the Study of Lung Distention in Relation to Circulatory Phenomena," *J. Lab. and Clin. Med.*, 4:556, 1919.
4 Drinker, P. and Shaw, L. A.: "An Apparatus for the Prolonged Administration of Artificial Respiration," *J. Clin. Investigation*, 7:229, 1929.
5 Gray, John S.: "Pulmonary Ventilation and Its Physiological Regulation," Springfield, Illinois, Charles C. Thomas, 1950.
6 Draper, W. B. and Whitehead, R. W.: "Diffusion Respiration in the Dog Anesthetized by Pentothal Sodium," *Anesthesiology*, 5:262, 1944.
7 Fenn, W. O.: "Mechanics of Respiration," *Am. J. Med.*, 10:77, 1951.
8 Maloney, J. V., Jr. and Whittenberger, J. L.: "Clinical Implications of Pressures Used in the Body Respirator," *Am. J. M. Sc.*, 221:425, 1951.
9 Barach, A. L., Fenn, W. O., Ferris, E. B. and Schmidt, C. F.: "The Physiology of Pressure Breathing. A Brief Review of Its Present Status," *J. Aviation Med.*, 18:73, 1947.
10 Barach, A. L., Martin, J. and Eckman, M.: "Positive Pressure Respiration and Its Application to the Treatment of Acute Pulmonary Edema," *Ann. Int. Med.*, 12:754, 1938.
11 Barach, A. L. and Swenson, P.: "Effect of Breathing Gases Under Positive Pressure on Lumens of Small- and Medium-sized Bronchi," *Arch. Int. Med.*, 63:946, 1939.
12 Maloney, J. V., Jr., Affeldt, J. E., Sarnoff, S. J. and Whittenberger, J. L.: "Electrophrenic Respiration. IX. Comparison of Effects of Positive Pressure Breathing and Electrophrenic Respiration on the Circulation During Hemorrhagic Shock and Barbiturate Poisoning," *Surg., Gynec. and Obst.*, 92:672, 1951.
13 Wolfe, E. F. and Murphy, D. P.: "Relief of Cardiac Dyspnea by Drinker Respirator," *J.A.M.A.*, 101:598, 1933.
14 Young, H. B. and Whittenberger, J. L.: "The Physiologic Basis of Therapy in Pulmonary Emphysema Accompanied by High CO_2 Levels," *J. Clin. Investigation*, 30:838, 1951.
15 Motley, H. L., Lang, L. P. and Gordon, B.: "Use of Intermittent Positive Pressure Breathing Combined with Nebulization in Pulmonary Disease," *Am. J. Med.*, 5:853, 1948.

Discussion

ALVAN L. BARACH, M.D., F.C.C.P.
New York, New York

The studies of Whittenberger and Maloney clearly indicated that mechanical ventilation of the lungs produced by increasing the negative pressure within the respirator produced physiologic effects that were quite similar to pressure breathing applied to the upper respiratory passage-way by means of a mask or hood. This contribution has been of considerable importance since artificial ventilation in the tank respirator may now be understood to have the consequences, both advantageous and disadvantageous, which result from so-called positive pressure respiration. (I have just tried the device which allows the patient to initiate the ventilation by a slight chest or diaphragmatic movement and it seemed comfortable and effective).

In our own studies 15 years ago, pressure breathing was found to produce a compensatory rise in venous pressure of approximately 40 per cent of the applied mean extra-pulmonary pressure in normal subjects and 70 per cent in patients with congestive heart failure; the circulation time was also markedly prolonged in the cases of cardiac insufficiency. The study of pressure breathing during the war, used in the development of methods to increase altitude tolerance of the individual, likewise revealed a consistent compensatory rise in venous pressure, with a decrease in cardiac output when high mean pressures were employed.

During the past two years Dr. Beck, Dr. Seanor and I have made comparative studies on the venous pressure response in human subjects to pressure breathing carried out by increasing the negative pressure within the tank and to pressure applied to the upper respiratory passage-way. Since the venous pressure response varies, as indicated above, observations on the same subjects were made with the two methods. No differences were found in these two methods of mechanical ventilation of the lungs in respect to this aspect of circulatory stress. Our studies, therefore, confirm the hypothesis of Whittenberger and Maloney. The consequences of increased air density in upper respiratory pressure create several changes that are of little or no clinical significance.

In studies which we have recently made on mechanically induced coughing, a swift opening valve was employed in order to allow a sudden outlet of air from the lungs, simulating the volume flow of air during a normal cough after a full inspiration. In the slide which I will show, the decrease in the mean pressure applied to the lungs is revealed when the swift opening valve is employed, namely, a decrease from 8 to 5 cm. In addition, the venous pressure rise is shown to be approximately one-half that which takes place with the conventional operation of the respirator, even when the same peak pressure is obtained. Since an adequate pulmonary ventilation is maintained with this type of mechanical respiration which simulates, to some extent, the human cough, its advantage in patients with impairment of the peripheral circulation is evident, namely, a decreased interference with the return of blood from the right heart. In conditions of pulmonary edema, a higher mean pressure may be desired, but in patients with peripheral circulatory failure, mechanical respiration with a sudden opening valve is to be preferred, either attached to the conventional respirator or to a pressure hood.

Dr. Whittenberger and Dr. Maloney have presented so many interesting contributions that I have had to select only one aspect of their valuable paper for discussion.

The Surgical Treatment of Pulmonic Valvular Stenosis*

WILLIAM H. MULLER, JR., M.D.
Los Angeles, California

Successful pulmonic valvulotomy for the relief of valvular pulmonic stenosis was first reported by Brock[1] in 1948. Sellors[2] reported one successful case almost simultaneously. Subsequent reports[3-8] have presented increasing evidence of the benefit this procedure offers patients who have valvular pulmonic stenosis.

Pulmonic stenosis may be associated with a variety of congenital cardiac vascular deformities. However, it most frequently occurs in the tetralogy of Fallot or as isolated pulmonic stenosis with an intact ventricular septum. The stenotic area may involve the valve itself, the infundibular area, or, occasionally, both. In the tetralogy of Fallot infundibular stenosis occurs much more frequently than the valvular type, while in pure pulmonic stenosis the infundibular type occurs rarely. Valvular stenosis lends itself to surgical correction much more easily than does infundibular stenosis. In the former type only incisions in the diaphragmatic valve are necessary, while in the latter type excision of muscular tissue at the point of narrowing is required.

Little is known concerning the etiology of pulmonic stenosis. It is established that rubella and other infectious diseases occurring during the first trimester of pregnancy increase the probability of congenital heart disease in the new born. Fetal endocarditis has been thought to be a major cause of valvular pulmonic stenosis; however, Allanby and Campbell[9] do not agree with this view. They feel that it does not explain the structure of the stenosed valve nor the frequent association of other congenital lesions with pulmonic stenosis.

In pure pulmonic stenosis cyanosis may or may not be present. Maude Abbott thought that cyanosis was due to capillary stasis, but this does not seem likely since cyanosis is absent in some patients who have the most marked degree of stenosis. Capillary stasis may be a contributory factor in the production of cyanosis

---

*Presented at the annual meeting of the California Chapter of the American College of Chest Physicians, May 12, 1951, Los Angeles, Calif.

From the Bay District Surgical Laboratory and the Department of Surgery, School of Medicine, University of California at Los Angeles.

Supported in part by a grant-in-aid from the United States Public Health Service.

when it occurs in older individuals as right ventricular failure ensues in the second or third decade of life. It is now known that the right to left shunt through the patent foramen ovale, which is present in approximately 70 per cent of these patients, accounts for the cyanosis. The intensity of cyanosis is determined for the most part, therefore, by the size of the foramen ovale and the degree of pulmonic stenosis. Cyanosis is generally not present at birth and frequently does not appear for several years. It may not appear until the latter part of the second or during the third decade of life. Whether or not it has been present before this period, it usually becomes more intense as disability increases. As right ventricular failure progresses, an increased amount of venous blood is shunted from the right auricle through the patent foramen ovale to the left auricle and increased cyanosis results.

The diagnosis of pulmonic valvular stenosis may be extremely difficult when it occurs in the tetralogy of Fallot. However, this problem is not encountered in pure pulmonic stenosis because the valvular type is nearly always present. Cardiac catheterization is usually of value only in making the diagnosis of obstruction in the region of the pulmonic valve and is not often of much aid in differentiating between the valvular and infundibular types. Angiocardiography may be of some assistance in this regard. As Brock and Campbell[10] point out, the stenosed infundibulum may be diagnosed by this technique if the stricture is not adjacent to the pulmonic valve. X-ray and fluoroscopic examination of the chest frequently gives the most information in regard to the diagnosis of valvular pulmonic stenosis. If there is poststenotic dilatation of the main pulmonary artery, one can be reasonably sure that pure valvular pulmonic stenosis is present. Occasionally, however, a high infundibular stenosis may also produce poststenotic dilatation. One can not be absolutely sure of the diagnosis until operation, at which time one can usually determine if there is valvular stenosis by palpation through the main pulmonary artery.

Most surgeons today use the method devised by Brock for dividing the stenotic pulmonic valve. This method consists of exposing the right ventricle through a left thorocotomy and passing a valvulotome through the wall of the right ventricle to engage the valve and divide it. Several different valvulotomes have been devised for this procedure. The original Brock valvulotomes are spoon shaped instruments which have a probe shaped tip on the leading end. The anterior edges of the cutting portion are sharp, while the lateral and posterior edges are blunt. Glover, Bailey and O'Neill[11] have modified this instrument so that it is malleable. More recently Potts[12] has devised a two bladed instru-

ment the blades of which can be opened to the cutting position after they enter the right ventricle, and we have devised a three bladed instrument similar to that of Potts (See Figure 1). The last two instruments have a slight advantage over the Brock valvulotome in that they can be introduced through a much smaller opening in the ventricle and less bleeding occurs. Brock originally used an instrument similar to a urethral sound to dilate the incised valve but more recently has devised an expandable dilator.[4] Similar dilating instruments have also been devised by Potts and by us.[8] Varco[13] has approached the problem in a different manner. He temporarily arrests the venous return to the heart by occluding the venae cavae. He is thus able to open the pulmonary artery and incise or excise a portion of the pulmonic valve under direct vision. He has used this method to excise the stenotic infundibulum in several cases of infundibular stenosis.

We have employed the Brock procedure in our patients. Initially we used only an expandable dilating instrument with the idea that tearing and dilating the pulmonic valve might be sufficient to relieve the stricture. However, because we could not be sure of

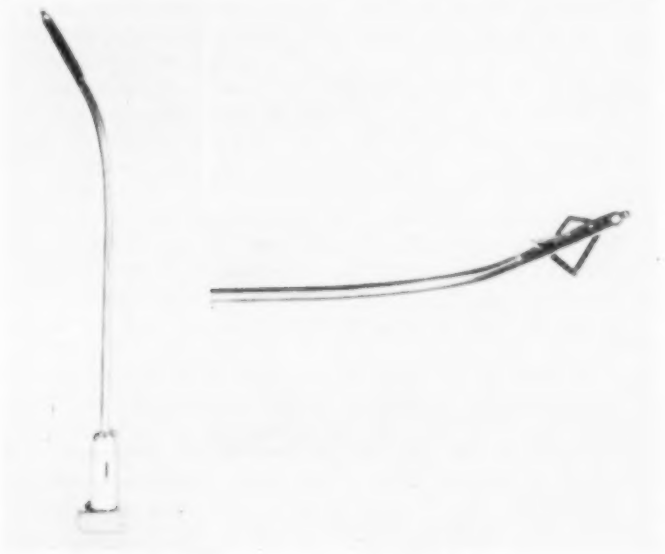


FIGURE 1a FIGURE 1b

Figure 1: Photographs of pulmonic valvulotome.
A. Overall view of instrument.
B. Close-up view of leading end showing blades in open position.

the manner in which the valve was treated by this instrument, we devised a three bladed cutting valvulotome, the blades of which could be expanded after they had been passed into the right ventricle.

The procedure we now employ is as follows: Penicillin is administered for about two days before operation, and in most instances Quinidine is given during this period to aid in reducing the occurrence of arrhythmias when the pericardium is opened. The patient is placed supine on the operating table, and the left shoulder is slightly elevated. The chest is entered through an anterolateral incision in the fourth intercostal space, and 5 to 10 cc. of 4 per cent novocain are placed in the pericardial cavity. The pericardium is opened anterior to the phrenic nerve, and the area about the infundibulum and the main pulmonary artery are palpated in order to determine the type of stenosis. If marked poststenotic dilatation is present, one can be reasonably sure that one is dealing with valvular stenosis, and the stenosed valve can usually be palpated through the pulmonary artery.

An area on the wall of the right ventricle 2 to 4 cm. proximal to the pulmonary artery is injected with 1 per cent novocain, and two stay sutures are placed in this area. A small stab wound is made between the stay sutures, and the valvulotome is introduced into the right ventricle through this opening (Figure 2). The blades

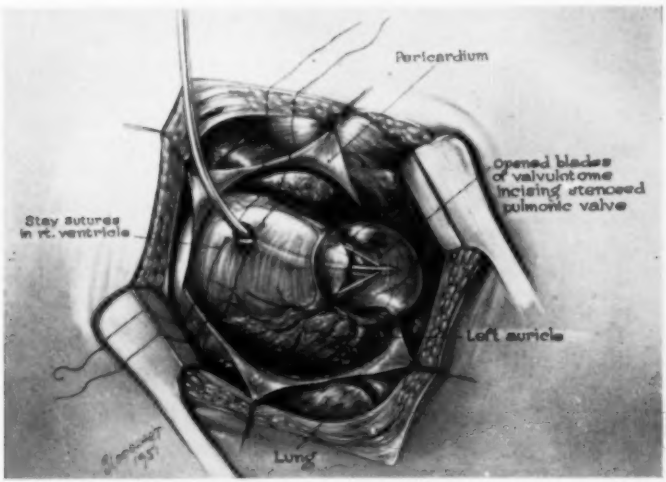


FIGURE 2: Drawing of operative exposure for pulmonic valvulotomy. The valvulotome has been introduced into the right ventricle, and the open blades are incising the stenotic pulmonic valve.

are then opened to the cutting position and the valvulotome is pushed through the stenosed pulmonic valve. The blades are closed, and the instrument is withdrawn. The expandable dilator is then passed into the ventricle (Figure 3), and the valves are dilated. During these manipulations arrhythmias often occur; however, they have not been significant. The opening in the right ventricle is closed with interrupted silk sutures, and the pericardium is closed loosely so that any fluid which accumulates in it may drain into the left pleural cavity. Intrapleural catheter drainage has been instituted in all our patients.

Discussion

Six patients with valvular pulmonic stenosis who were operated on by the Brock method have benefited. Cyanosis was present in two before operation. After operation it markedly diminished in one and disappeared in the other. Two had ventricular septal defects. The exercise tolerance in all of these patients has increased. The systolic murmurs which were present before operation still persisted after valvulotomy but were of much less intensity. Diastolic murmurs did not appear in any of the patients.

A seventh patient who was thought to have valvular stenosis with an interventricular septal defect was found at operation to have low infundibular stenosis. An infundibular chamber was present distal to the point of stenosis and was mistaken for post-stenotic dilatation of the main pulmonary artery during the

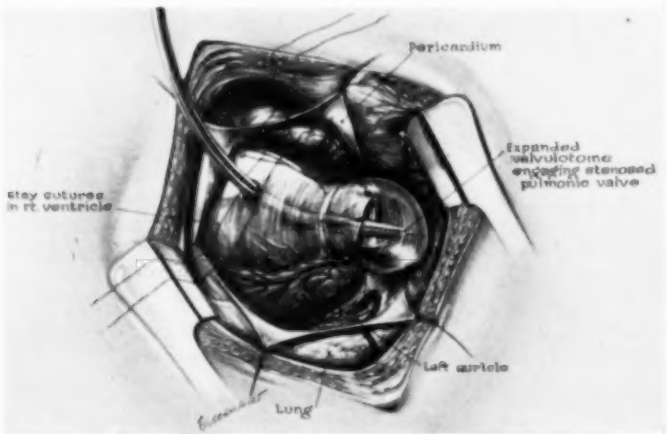


FIGURE 3: Drawing of operative exposure for pulmonic valvulotomy. The incised valve is being dilated by the expansible dilator.

preoperative x-ray and fluoroscopic examination. The stenosis in this patient was of the diaphragmatic type and was divided with the pulmonic valvulotome. Although the patient appears to be improved, insufficient time has elapsed since the operation to permit proper evaluation.

Recently Brock and associates[10] and Glover and his co-workers[11] have treated patients with the tetralogy of Fallot by relieving the point of obstruction. Since infundibular stenosis occurs in a high percentage of these cases, resection of the stenotic infundibulum has been necessary. They state that the reason for using the direct method is to reduce the number of defects rather than to add another, as is done when a systemic-pulmonary artery anastomosis is performed. A reduction in the number of defects is highly desirable, but unless the direct method results in a mortality rate as low as that of the shunt method, the latter one should be used.

The method which Varco has developed for pulmonic valvulotomy is a logical approach to the problem. One would think that more adequate incisions could be made in the valve under direct vision. We have divided the pulmonic valve in dogs under direct vision by temporarily occluding the outflow tract of the right ventricle with a Potts ductus clamp. This method has not been used in patients with pulmonic stenosis but might be applicable when an interventricular septal defect is present so that the right ventricle may empty through it during the period of occlusion.

SUMMARY

A method for pulmonic valvulotomy has been devised by Brock and employed successfully by other surgeons.

Instruments which we have devised and which are used for the Brock procedure are described.

Seven cases of pulmonic stenosis in which the Brock procedure was performed are discussed.

RESUMEN

Brock ha ideado un método de valvulotomía pulmonar, y se ha usado con éxito por otros cirujanos.

Se describen los instrumentos que se han ideado y que son usados en el procedimiento de Brock.

Se discuten siete casos de estenosis de la pulmonar en los que el procedimiento de Brock se realizó.

RESUME

Une méthode de valvulotomie de la pulmonaire a été décrite par Brock et utilisée avec succès par d'autres chirurgiens.

L'auteur décrit les instruments qu'il a fait construire et ceux

qui sont utilisés pour le procédé de Brock. Il rapporte sept cas de sténoses de l'orifice pulmonaire dans lesquels à été utilisée la technique de Brock.

REFERENCES

1 Brock, R. C.: "Pulmonary Valvulotomy for Congenital Pulmonary Stenosis," *British M. J.*, 1:1121, 1948.
2 Sellors, T. H.: "Surgery of Congenital Heart Disease," *Lancet*, 2:71, 1948.
3 Brock, R. C.: "Surgery of Pulmonary Stenosis," *British M. J.*, 2:399, 1949.
4 Brock, R. C. and Campbell, Maurice: "Valculotomy for Pulmonary Valvular Stenosis," *British Heart Jour.*, 12:377, 1950.
5 Blalock, Alfred and Kieffer, Richard F.: "Valvulotomy for the Relief of Congenital Valvular Pulmonic Stenosis with Intact Ventricular Septum," *Annals of Surgery*, 132:496, 1950.
6 Potts, Willis J., Gibson, Stanley, Riker, William L. and Leininger, C. R.: "Congenital Pulmonary Stenosis with Intact Ventricular Septum," *J.A.M.A.*, 144:8, 1950.
7 Muller, William H., Jr. and Longmire, William P., Jr.: "The Surgical Treatment of Cardiac Valvular Stenosis," *Surgery*, 30:29, 1951.
8 Muller, William H., Jr. and Longmire, William P., Jr.: "The Surgical Treatment of Pure Pulmonic Stenosis: A Description of an Expansible Valvulotome," *Surgery*, 30:275, 1951.
9 Allanby, K. D. and Campbell, Maurice: "Congenital Pulmonary Stenosis with Closed Ventricular Septum," *Guy's Hospital Reports*, 98: 18, 1949.
10 Brock, R. C. and Campbell, Maurice: "Infundibular Resection or Dilatation for Infundibular Stenosis," *British Heart Jour.*, 12:403, 1950.
11 Glover, Robert P., Bailey, Charles P. and O'Neill, Thomas J. E.: "The Direct (Brock) Relief of Pulmonary Stenosis in Tetralogy of Fallot," Read before the 31st Annual Meeting of The American Association for Thoracic Surgery, April 16-18, 1951, Atlantic City, New Jersey.
12 Potts, W. J.: Discussion of paper, "The Direct (Brock) Relief of Pulmonary Stenosis in Tetralogy of Fallot," Glover, Robert P., Bailey, Charles P. and O'Neill, Thomas J. E., Read before the 31st Annual Meeting of The American Association for Thoracic Surgery, April 16-18, 1951, Atlantic City, New Jersey.
13 Varco, Richard L.: Discussion of paper, "The Surgical Treatment of Cardiac Valvular Stenosis," Muller, William H., Jr. and Longmire, William P., Jr., *Surgery*, 30:29, 1951.

Boeck's Sarcoid: Observations on the Use of BCG Vaccine*

MARVIN S. HARRIS, M.D., F.C.C.P.† and CHARLES SHORE, M.D.††
Los Angeles, California

Sarcoidosis may be defined as a non-caseating granulomatous reaction to an unknown etiologic agent or agents. In all probability, the nature of the host's reaction is more important than the specificity of the causative factor. Evidence has been offered that this factor may be the tubercle bacillus,[1] but proof of this is not conclusive, and other causes have not been excluded.

The behaviour of the disease is extremely variable in terms of anatomical distribution and clinical course.[2] The latter may be acute or chronic, relentlessly progressive or remittent. By some it is regarded as a benign disease, by others as a severe threat to life. The wide limits of clinical behaviour therefore make difficult a critical evaluation of attempts to modify the course of the disease.

BCG vaccination has been employed to prevent the later development of clinical tuberculosis in those cases of sarcoidosis anergic to tuberculin, as a possible means of diagnosis,[3] and finally for therapeutic effects.[4] The usefulness of these efforts has neither been fully established, nor conclusively disproved.

In exploring the therapeutic effect of BCG, we observed, as others have, the fairly frequent failure to secure a typical dermal reaction to the vaccine. Leider and Sulzberger, for example, in vaccinating four anergic cases of sarcoidosis, noted the height of the reaction to occur in five to seven days, with the appearance of non-ulcerating indolent papules. This is in contrast to the normal individual, who reacts to intracutaneous administration of BCG with erythema, papulovesiculation, and eventual scar formation—the height of the reaction occurring in two weeks.[5] Of even greater significance is the frequent failure to obtain tuberculin skin sensitivity following vaccination in sarcoid patients, and failure to sustain such sensitivity when it does develop.

The possibility that hyaluronidase might influence the intensity of the vaccine reaction—or "take"—and thus enhance the development of skin sensitivity was suggested. Duran-Reynals demon-

---

*Paper read at meeting of California Chapter of The American College of Chest Physicians, May 12, 1951, Los Angeles, California.

†Senior Attending, Tuberculosis Service, Los Angeles County General Hospital.

††Resident Physician, Tuberculosis Service, Los Angeles County General Hospital.

strated that hyaluronidase increased the intensity of local infection without altering the virulence of the infecting organism.[6] Israel, et al., failed to note any correlation between the severity of the BCG vaccine reaction and the development of tuberculin sensitivity in sarcoid patients.[7]

Seven patients with sarcoidosis proved by clinical course and biopsy, and anergic to tuberculin, were studied. BCG provided by Dr. S. R. Rosenthal of the Tice Laboratories was used, and the multiple puncture technique was employed. Fifteen of the recommended 30 punctures were made over an 0.1 cc. saline wheal in the skin, the remaining 15 over a wheal in which 0.1 cc. of saline contained 100 units of freshly prepared hyaluronidase.

All patients were skin tested shortly before the vaccination with .005 mgm. PPD. The vaccination sites were inspected at frequent intervals, and skin tests with PPD repeated at monthly intervals. Successful vaccination was indicated by the appearance of inflamed nodules in the skin, reaching the peak of reaction between seven and 14 days. No instance of frank ulceration was observed.

In two of the patients, the only distinctive visible reaction to the vaccine was noted over the hyaluronidase wheal. In four others, the reaction over the hyaluronidase wheal was unquestionably more intense at two weeks. By four weeks, no discernible difference could be detected. In the seventh patient, both vaccinations were of equal intensity. (This patient became a reactor to tuberculin).

Sensitivity to tuberculin developed in *five* of the *seven* patients. Of these five, sensitivity disappeared in two months in two, and decreased in a third patient. While the incidence of "takes" is somewhat higher than recorded by other observers, the number of cases studied prohibits any statistically significant statement.

The rapid decline of tuberculin allergy in more than one-half of the initially positive reactors is in general accord with the findings of Israel.

As to the therapeutic effects, observations have been made on these seven, and three additional patients. Four observed marked improvement in general health, and subsidence of cough and dyspnea. No change was noted in the physical or x-ray findings, so that factors of coincidental remission and suggestion cannot be eliminated. A fifth patient with hepatic and splenic involvement and signs of portal obstruction, critically ill at the time of vaccination, improved in a short time to a most unexpected extent. However, the fact that skin lesions progressed shortly after the period of improvement suggests a spontaneous alteration in the course of the disease, rather than an effect of the vaccination, which, incidentally, did not produce tuberculin skin sensitivity.

In two patients who received .005 mgm. PPD 48 hours before vaccination, with a negative response, there was observed one week after vaccination, definite erythema and induration at the site of the previously negative skin test. The lapse of one week following vaccination is insufficient for the development of tuberculin skin sensitivity and subsequent skin test on these patients failed to disclose comparable degrees of tuberculin skin reaction. This suggests that these anergic patients had a latent tuberculin allergy which was activated by the introduction of viable antigen in the form of BCG. The relationship of this phenomenon to a possible tuberculous etiology of Boeck's Sarcoid provides an interesting though inconclusive basis for further speculation.

SUMMARY

1) Local injection of hyaluronidase is capable of intensifying the reaction to the multiple puncture application of BCG vaccine.

2) The intensification of this reaction apparently has no influence on the persistence of tuberculin skin sensitivity. Its effect on the *development* of such sensitivity is questionable.

3) Therapeutic value of BCG vaccination cannot be completely ruled out, but appears unlikely.

4) A late flare-up at the site of previously negative tuberculin skin test following BCG vaccination is reported and its possible significance suggested.

RESUMEN

1) Una inyección local de hyaluronidasa es capaz de intensificar la reacción a la aplicación por punción múltiple de la vacuna B.C.G.

2) La intensificación de esta reacción parece no tener influencia alguna sobre la persistencia de sensibilidad de la piel a la tuberculina. Su efecto en el *desarrollo* de tal sensibilidad es dudoso.

3) El valor terapeutico de la vacunación con B.C.G. no puede ser desechado, pero parece poco probable.

4) Se informa de una erupción tardía en el sitio donde una prueba con tuberculina hecha con anterioridad había resultado negativa después de la vacunación con B.C.G., y se sugiere el significado que posiblemente implique.

RESUME

1) L'injection locale de hyaluronidase est capable d'augmenter la réaction des scarifications pratiquées pour vacciner par le B.C.G.

2) L'intensification de ces réactions n'a aucune influence apparente sur la persistance de la sensibilité cutanée à la tuberculine. Son effet sur le développement de cette sensibilité peut être mise

en doute. L'action thérapeutique du B.C.G. ne peut pas être complètement exclue mais parait improbable.

4) Les auteurs rapportent la possibilité d'une apparition tardive des réactions tuberculiniques cutanées au lieu même où antérieurement ces réactions étaient restées négatives après vaccination par le B.C.G. Ils proposent une explications de ce phénomène.

REFERENCES

1 Pinner, M.: "Noncaseating Tuberculosis: Analysis of Literature," *Amer. Rev. Tuberc.*, 37:690, 1938.
2 Riley, E. A.: "Boeck's Sarcoid," *Amer. Rev. Tuberc.*, 62:231, 1950.
3 Leider, M. and Sulzberger, M. B.: "Studies in the Allergy of Infection: Responses of the Skin to BCG Vaccination in Various Categories of Tuberculin Sensitivity," *Jour. Invest. Derma.*, 13:249, 1949.
4 Cornbleet, T. and Rosenthal, S. R.: Discussion of Leiden, M. and Sulzberger, M. B., *Jour. Invest. Derma.*, 13:249, 1949.
5 Leider, M. and Sulzberger, M. B.: "Studies in the Allergy of Infection: Responses of the Skin to BCG Vaccination in Various Categories of Tuberculin Sensitivity," *Jour. Invest. Derma.*, 13:249, 1949.
6 Duran-Reynals, F.: "Tissue Permeability and the Spreading Factors in Infection," *Bacter. Reviews*, 6:197, 1942.
7 Israel, H. L., Sones, M., Stein, S. C. and Aaronson, J. D.: "BCG Vaccination in Sarcoidosis," *Amer. Rev. Tuberc.*, 62:408, 1950.

Delayed Reactions to Bouillon Medium of Tuberculin*

M. R. LICHTENSTEIN, M.D., F.C.C.P.† and C. A. CILELLA, M.D.††
Chicago, Illinois

In testing persons who are negative to weak dilutions of old tuberculin, it is frequently noted that a disproportionately large reaction may occur with the stronger dilutions. The suspicion has been voiced that these reactions to strong concentrations of old tuberculin may be non-specific. The problem is of special importance in testing children prior to BCG administration. Are those who react only to a 1:100 or 1:10 strength of old tuberculin to be looked upon as previously infected and not in need of BCG, or is this a non-specific reaction, not indicative of previous exposure?

Delayed reactions to old tuberculin, not caused by the tuberculoprotein per se, are theoretically possible from two causes principally:

1) Irritation of injected materials due to acidity or alkalinity, excessive concentration of salts, or chemicals causing an inflammatory response.

2) Allergic responses to substances derived from the bouillon medium.

This problem has been attacked by a number of workers. Aronson[1] reported on sensitization to MA100; Furcolow[2] on reactions to purified protein derivative in infants; Palmer[3] on the geographic distribution of non-specific reactions; and Hart[4] and Freund and Hart[5] on reactions to substances in the medium.

This report concerns an investigation of hypersensitivity to bouillon medium.

Materials and Methods

Old Tuberculin (Parke, Davis and Company) diluted in a buffered diluent[6] was used in the following strength: 1:10,000, 1:1,000, 1:100, and 1:10. Tuberculin PPD was used in the second strength and also the same diluted 1:10 and 1:100.

The bouillon medium generously furnished by Parke, Davis and Company was made in a manner identical with old tuberculin

---

*From the Municipal Tuberculosis Sanitarium, Chicago, Illinois.
†Chief of Medical Service, Municipal Tuberculosis Sanitarium.
††Staff Physician, Municipal Tuberculosis Sanitarium.

Presented at the 17th Annual Meeting of the American College of Chest Physicians, Atlantic City, New Jersey, June 7-10, 1951.

except that it was not inoculated with tubercle bacilli. The formula is as follows: Peptone 2 per cent, sodium chloride 0.5 per cent, glycerin 5 per cent, and beef infusion Q.S. The P.H. is adjusted to 7.6. The medium is sterilized at high steam pressure. The sterile bouillon is then evaporated over a steam bath to 1/10 the original volume. This batch was prepared in September 1949. This material was used in the same dilutions and diluent as old tuberculin (freshly made bouillon was tried, but was nonreactive).

A preparation of Long's medium was made and concentrated 10 times over a steam bath. It was used in a 1:10 dilution.

New glass was used for the bouillon and Long's medium.

All tests were done by the Mantoux method, using 0.10 cc. intracutaneously. The reactions were read at 72 hours and the transverse diameter of the indurated area was recorded. In one group the duration of the induration was studied by following it daily until it disappeared.

Tests were performed on the following groups of persons:

1) 34 tuberculous children.
2) 426 tuberculous adults.
3) 150 newly hired employees.
4) 88 affiliate student nurses.

The latter group came from a rural area, thus providing individuals who were completely insensitive to tuberculin.

Reactions were considered negative if there was no induration, doubtful if there was induration up to 5 mm. in diameter, and positive if over 5 mm. in diameter.

Control tests with diluent alone established the fact that this material caused no reactions.

The large majority of tests were done by giving simultaneous injections of old tuberculin 1:10,000 and bouillon. The latter was usually the 1:10 dilution. Tests with bouillon 1:100 and 1:1000 however were also carried out. All of the tuberculous patients had received tests with old tuberculin during the preceding several months.

Sixteen patients who were negative to the first two tests with bouillon 1:10 received 10 successive intracutaneous tests each at 48-hour intervals.

Seventy-four of the affiliate nurses were completely insensitive to tuberculin in four strengths including 10 mg. of old tuberculin. This series of tests was repeated at the end of two months and tests with bouillon 1:10 were included at the beginning and end of each series.

A number of variations of the above experiments were carried out. Some of the patients were tested with PPD instead of old tuberculin.

Results

Tests with bouillon medium gave positive reactions at 72 hours which were indistinguishable by inspection and palpation from the usual tuberculin reaction. The reactions were sometimes very firm and indurated and at others soft and puffy. They ranged in size up to 2 cm. in diameter of the induration. Additional erythema was frequent. Bleb formation occurred in two patients.

Observations on the duration of these reactions indicated that they last as long as 10 days, depending upon their size and intensity. They usually did not remain quite as long as the tuberculin test which was put on the skin simultaneously.

The large majority of the reactions were obtained with a 1:10 dilution of the bouillon. Forty-four per cent of the adult patients and 20 per cent of the tuberculin positive employees tested gave definitely positive reactions to this material. A small percentage gave doubtful reactions and the remainder were completely negative.

Tests with bouillon 1:100 and 1:1000 were positive quite frequently among the children. Fifty per cent of the tuberculous children reacted to the 1:100 dilution as compared to 16 per cent of adults. Forty per cent of the tuberculous children reacted to

TABLE I

Reactivity to Bouillon of Various Groups Tested

	Number of Patients	Reaction to Tuberculin	Percentage Positive to Bouillon 1:10	Percentage Positive to Bouillon 1:100	Percentage Positive to Bouillon 1:1000
Student Nurses	88	14 positive	28		
		74 negative	0		
Employees	150	18 negative	0		
		39 positive	17		
		57 positive (not previously tested)	15		
		36 positive (previously tested	33		
Tuberculous Children	34	24 positive		50	
		10 positive			40
Tuberculous Adults	426	307 positive	44		
		86 positive		16	
		33 positive			12
Tuberculous Adults Over 60 years old	18	positive	55		

the 1:1000 dilution as compared to 12 per cent of the adults. Reactions to the 1:10,000 dilution were either negative or doubtful. (The total number of children tested [see Table I] was too small to be statistically significant.)

Tests were performed on a series of moribund patients who required a 1:10 dilution of old tuberculin to react. Ten of these were simultaneously tested with 1:10 bouillon and it was found that the reaction to the bouillon was about two-thirds the size of the reaction to old tuberculin.

Eighty-eight affiliate student nurses were tested, of whom 14 were tuberculin positive. Four of these were positive to bouillon 1:10. Of 74 tuberculin negative nurses, none reacted to bouillon. Thirty-four of these nurses received 12 injections each of old tuberculin and bouillon over a two-month period, but remained completely negative to both.

The 16 tuberculin positive patients who received 10 successive injections of 1:10 bouillon all remained negative to this material.

Of 36 tuberculin positive employees who stated that they had previously had tuberculin tests, 33 per cent were positive to the bouillon. Of 57 similar employees who stated that they had not been previously tested, 15 per cent were positive. Eighteen tuberculin negative employees did not react to bouillon. Retests two to 12 weeks later revealed that some of those negative at first became positive to the bouillon later.

Tests with Long's medium were uniformly negative, including patients repeatedly tested with PPD.

Discussion

It is evident from the above results that the bouillon medium is capable of giving reactions very similar to tuberculin. Strong concentrations are required, usually 1:10, although sometimes 1:100 or 1:1000 react. No reaction was observed to this material among non-reactors to tuberculin. Among those sensitive to tuberculin, a considerable proportion react to the bouillon.

It is apparent moreover that this reaction is not due to irritation, since it is absent among non-reactors to tuberculin. It is an allergic response to a specific substance in the bouillon and is limited to a portion of the tuberculin reactors. The specific substance is probably beef protein, perhaps altered chemically during the process of concentration over the steam bath, by aging, or by combination with one or more of the other ingredients of the medium.

Many of the tuberculin reactors who were negative to the bouillon could not be sensitized to the latter even after numerous intradermal injections. We were unable to note any basic dif-

ference otherwise between those who were sensitive to bouillon and those who could not be sensitized.

It appears probable that hypersensitivity to bouillon is artificially produced by the injection of old tuberculin (which of course contains bouillon). This follows from the fact that repeated tests of various groups produced successively larger percentages of reactors to bouillon. Also, employees who stated that they had previously had tuberculin tests gave a higher percentage of positives to bouillon than those not previously tested. Although some of the latter group were positive to bouillon, we assume that they may have forgotten a previous test, perhaps performed when they were children.

Since many reactors to old tuberculin do not react to bouillon, it is evident that sensitivity to the latter does not depend upon cross-reactivity, that is, a chemical similarity between bouillon and tuberculoprotein.

Raffel[7] has demonstrated that sensitization to tuberculoprotein and other substances can be accomplished in the non-tuberculous animal by using the wax from the tubercle bacillus as an adjuvant. The presence of the wax apparently is necessary to establish the delayed type of hypersensitivity. The absence of this wax from the bodies of those who have never had a primary infection with tubercle bacilli may be the reason it is impossible to artificially sensitize them with the bouillon. To explain our inability to sensitize some of the tuberculous patients we may speculate that the wax, though present in their bodies, is unavailable because it is chemically bound or changed.

Testing with serial dilutions of old tuberculin thus introduces the possibility that an increasingly large portion of the reaction is due to the bouillon rather than to the tuberculoprotein. Even though this is true, such reactors are definitely tuberculin positive because, as noted above, non-reactors are invariably negative to the bouillon. This means that those children and others being tested prior to BCG inoculation who give delayed but so-called non-specific reactions to old tuberculin in the stronger concentrations probably must be considered reactors to tuberculin and, therefore, do not require BCG.

We believe that hypersensitivity to bouillon accounts for many, but not all, so-called delayed non-specific reactions to old tuberculin.

When sensitivity to bouillon is present, it seems to increase following repeated injections of either old tuberculin or bouillon. If this is true, then the anamnestic reaction to old tuberculin will require reinvestigation to determine whether it is due to the bouillon or the tuberculoprotein.

In the case of PPD, which is made by growing tubercle bacilli on Long's medium, the non-specific reactions which have been reported must be explained on some other basis, since we were unable to obtain any reactions with Long's medium.

Thus, it appears that the medium of growth plays an important role in the production of reactions to old tuberculin but not PPD.

SUMMARY

1) Certain preparations of bouillon medium, but not all, are capable of eliciting delayed reactions very similar to those of old tuberculin.

2) These reactions do not occur in persons completely insensitive to tuberculin. They occur in a considerable proportion of, but not all, tuberculin reactors.

3) Those tuberculin reactors who were negative to bouillon on the first two tests could not be sensitized by multiple injections.

4) Preparations of Long's medium did not produce delayed reactions.

5) The theoretical implications and possible practical applications of these findings are discussed.

Acknowledgment: We are very appreciative of the help of Miss Wilma Cannemeyer, medical technician.

RESUMEN

1) Ciertas preparaciones por medio de caldo son susceptibles de provocar reacciones similares retardadas muy similares a las de la tuberculina antigua.

2) Esas reacciones no ocurren en personas completamente insensibles a la tuberculina. Acontecen en una considerable parte de los reactores a la tuberculina, pero no en todos.

3) Los reactores de tuberculina que fueron negativos al caldo en las dos primeras pruebas, no pudieron ser sensibilizados por inyecciones múltiples.

4) Las preparaciones del medio de Long no produjeron reacciones retardadas.

5) Las consecuencias teóricas y probablemente prácticas de estos hallazgos son discutidas.

RESUME

1) Certaines préparations de bouillon de culture, et non toutes, sont susceptibles de créer des réactions tardives, absolument comparables à celles que peut déterminer la tuberculine brute.

2) Ces réactions ne surviennent pas chez des gens qui sont

parfaitement insensibles à la tuberculine. Elles apparaissent dans une proportion considérable, mais non pas exclusive, chez des individus allergiques.

3) Les individus allergiques qui ne réagissaient pas au simple bouillon ne purent être sensibilisés par des injections multiples.

4) Les préparations faites sur le milieu de Long ne provoquent pas de réactions tardives.

5) Les auteurs discutent les applications théoriques et pratiques de ces constatations.

REFERENCES

1 Aronson, J. D. and Nicholas, R. V.: "The Comparative Value of Tuberculoprotein MA100 and Old Tuberculin with a Special Reference to Sensitization," *J. of IMM.*, 25:483, 1933.

2 Furcolow, M. L., Hewell, B., Nelson, W. E. and Palmer, C. E.: "Quantitative Studies of Tuberculin Reaction," *Pub. Health Reports*, 56:1082, 1941.

3 Palmer, C. E., Ferrebee, B. A. and Petersen, O. S.: "Geographic Differences in Sensitivity to Tuberculin as Evidence of Nonspecific Allergy,"*Pub. Health Reports*, 65:1111, 1950.

4 Hart, P. D'A.: "Specificity of Skin Reactions in Man in Relation to Tuberculous Infection," *Quart. J. of Med.*, (Oxford), 10:49, 1932.

5 Freund, J. and Hart, P. D'A.: "Skin Reactivity to Glycerinated Veal Broth and It's Bearing on Specificity of Tuberculin Reaction," *Quart. J. of Med.* (Oxford), 28:417, 1935.

6 Gottschall, R. and Bunney, Wm. E.: "Diluent for Stabilizing Old Tuberculin," *J. of IMM.*, 34:103, 1938.

7 Raffel, Sidney and Forney, J. E.: "The Wax of the Tubercle Bacillus in Hypersensitivity to Picryl Chloride," *J. of Exp. Med.*, 88:485, 1948.

Discussion

E. A. PISZEK, M.D., F.C.C.P.
Chicago, Illinois

Doctors Lichtenstein and Cilella are to be congratulated on their original presentation regarding delayed reactions.

Certainly the specificity of the tuberculin reaction once considered to be absolute continues under scrutiny and the observations of Dr. Lichtenstein and Cilella warrant further work to determine, if possible, the reason for the reaction to the bouillon medium. It may be noted that non-specific reactions are seen in other forms of skin testing. People who have previously had Schick or Dick tests may subsequently develop false positives on re-testing in the same skin area apparently due to local allergic sensitivity.

When Schick tests became popular, it was first recommended that they be read in 48 hours. It was soon learned that a high percentage of positive reactions were false positives; now Schick

tests are read a week following the test and false positives are eliminated by this delay in reading. This, of course, is not comparable to the tuberculin reaction but probably does represent an induced local hypersensitivity which is not possible with tuberculin. In view of this I would like to ask if multiple or single sites were used to attempt to sensitize the negative bouillon reactors. Also of interest are the groved cases of tuberculosis with a negative tuberculin reaction as recently re-stated by Mascher in the American Review of Tuberculosis for May, 1951.

I hope that the authors will continue to interest themselves in the tuberculin reaction as they have done for many years.

Lingular Disease

WILLIAM A. HOPKINS, M.D., F.C.C.P.* and TED F. LEIGH, M.D.*
Atlanta, Georgia

Since the introduction of segmental resection of the lung by Churchill and Belsey[1] in 1939, considerable interest has focused itself upon the treatment of solitary segmental disease of the lung.

The relationship of lingular bronchiectasis to left lower lobe bronchiectasis has been known for a long time and has been aptly and completely discussed by such authors as Churchill and Belsey, Overholt and Langer, and Blades and Kent.[1-3]

It has been shown that bronchiectasis of the lingula associated with left lower lobe bronchiectasis occurs in between 75 and 80 per cent of cases. In contrast, the presence of solitary lingular bronchiectasis without segmental disease of the lung elsewhere has been infrequent. Churchill[1] found only four in 50 cases with bronchiectasis. Schmidt[4] reported his findings in 555 bilateral bronchograms of which 257 had positive findings. Investigation of this positive group revealed evidence of non-tuberculous bronchiectasis in 158. Of these, solitary lingular disease was found in only two instances. Overholt, Betts and Woods[5] made no mention of the occurrance of isolated lingular bronchiectasis in 39 patients who had had 53 separate operations. Clagett and Deterling[6] in 1946, presented their findings on the technic for segmental pulmonary resection, with special reference to associated lingular and lower lobe disease. They did not record any case of solitary segmental lingular bronchiectasis.

We have encountered 32 patients having solitary lingular disease of whom 15 had bronchiectasis. These 15 are from a total of 370 cases with bronchiectasis seen at this clinic. Thus one can surmise that this entity is not as rare as would be considered from the above reports. Our experience has been essentially that of other authors relative to the combination of lingular bronchiectasis associated with lower lobe disease on the left side.

There are other diseases which occur within the lingular segment of the left upper lobe which are of equal importance. The lingula is not unique in its particular disease entities, as these same processes are found scattered throughout the lung.

In most instances lingular disease may be identified on routine

*From the Departments of Surgery and Radiology, Emory University School of Medicine, Atlanta, Georgia, and Emory University Hospital, Emory University, Georgia.

posterior-anterior and lateral roentgenograms of the chest without the aid of more elaborate diagnostic procedures. It is in the recognition of the signs, symptomatology, and roentgenological appearance of lingular disease that this paper is particularly applicable.

The term *lingula* refers to the tip or tongue-like projection of the upper lobe of the left lung but in general it is considered also to be the entire portion of this segment which is supplied by the first segmental bronchus that arises from the upper lobe bronchus.

Failure to recognize this as a separate segmental entity has resulted in a considerable number of failures in the treatment of bronchiectasis involving the left lower lobe. The upper margin of the lingula is often demarcated by a fissure or indentation in the anterior margin of the upper lobe. This extends in varying degrees of depths into the upper lobe, and in some cases it may be complete, thereby separating the left lung into three major lobes.

The detailed anatomy of the arrangements of the parts of the lung was first described by Ewart[7] and in general has remained essentially the same except for some change in nomenclature. The lingular division of the left upper lobe has been described in detail by many authors including Boyden, Jackson and Huber, and Foster-Carter and Hoyle.[8-10] There are only minor differences in the anatomy described by these authors. In general, they describe two major divisions which arise from a single bronchus, and this is usually the first division of the left upper lobe bronchus; it is the most inferior branch of this bronchus. Occasionally the upper lobe bronchus may bifurcate into an apical-posterior trunk and an anterior trunk. In these cases the lingular bronchus arises from the latter. The main lingular trunk soon divides into two branches called, by Ewart,[7] the cardiac and the outer mammary branches. These same segments were named by Boyden[8] and Jackson and Huber[9] as the superior and inferior lingular segments, or, if we wish to number them according to Jackson and Huber,[9] segments IV and V of the left lung. The superior lingular segmental bronchus soon divides into an anterior and posterior division, and the inferior lingular bronchus bifurcates into two parallel divisions. Most authors consider the lingula to be equivalent to the right middle lobe and have adopted, in general, the same type of description and nomenclature.

The upper branch of the lingula as it gives off its lateral or axillary branch has been considered by some workers to be a separate segment, and this axillary or lateral branch may often be the site of disease alone. However, the inferior division (segment V of Jackson and Huber[9]) is commonly the most involved segment. In all our cases both segments have been involved.

As described by Ewart, Blades and Kent, and others,[7,3,11] the arterial supply to the left upper lobe is extremely variable, and there may be anywhere from three to nine segmental branches. However, the location as well as the number of branches to the lingular segment is less variable than are the other branches of the upper lobe. Usually the arteries arise from a common trunk. It is important to realize that this common trunk often leaves the main pulmonary artery distal to or at a level with the branches to the lower lobe. In all of the cases encountered here the lingular artery was distal to the superior division of the lower lobe. Boyden[11] feels that the lingular artery usually arises as a single trunk. Occasionally both segmental bronchi may arise separately from the pulmonary artery, or they may arise from the anterior segmental artery. Knowledge of this unusual arrangement is important in resection of the lingula because the artery to the anterior segment must not be disturbed if the function of that segment is to remain.

Pulmonary veins are, in all cases, more variable than arteries in regard to the segmental division of the lungs, and this is true of lingular veins. Usually the upper lobe vein on the left side divides into three major trunks, the most inferior of which is the lingular division. However, the lingular division may also come from the anterior trunk and in some cases, as described by Boyden,[11] may even be a branch of the inferior pulmonary vein. The lingular vein itself may divide into one or two segmental veins early, or may extend far out into the substance of the lingula before it divides. The importance of intersegmental veins has been described by Overholt and Langer[2] in relation to segmental resection of the lung. In the case of the lingular segment, it is felt by Overholt that the presence of an intersegmental vein is not constant.

Clinical Picture

Patients with lingular disease present some variability in their symptomatology, but the clinical picture is rather typical in most instances. The group of cases with bronchiectasis presents a rather characteristic picture which, in the majority of instances, leads one to suspect the diagnosis from the history and physical findings. Chart I shows the symptomatology presented by this group of 32 patients with lingular disease. In all but five cases in this series the diagnosis was confirmed by operation or bronchogram. In this series there were four cases of carcinoma of the lung, and they will be discussed in detail later in this paper. There were two cases of chronic interstitial pneumonitis both proved by operation and pathological examination of the removed segment. There

was a chronic granuloma in one case and an abscess in the lingula in another. There were two instances of so-called atypical pneumonia. The diagnosis was assumed in these latter cases by the patient's clinical course and positive cold agglutinins. There was one pneumonococcal pneumonia which had a positive sputum for the pneumococcus organism and responded rather rapidly to chemotherapy. There are five cases in this series who had the clinical picture and roentgenological appearance on their x-ray films of having lingular disease, but they were not operated on

CHART I
SYMPTOMATOLOGY

	Bronchiectasis (15 cases)	Carcinoma (4 cases)	Others (13 cases)
Cough	15	4	10
Wheeze	7	3	6
Dyspnea	8	4	4
Fatigue	11	4	6
Hemoptysis:			
Slight	1	2	4
Severe	3	1	1
Chest pain	7	4	7
Fever	12	3	9
Sputum production:			
Moderate	11	2	7
Severe	3	1	0
Weight Loss	3	2	2

CHART II
DIAGNOSES

Diagnosis	No. of Cases
Bronchiectasis	15
Carcinoma	4
Diagnosis Undetermined	5
Atypical Pneumonia	2
Tuberculosis	1
Chronic interstitial pneumonitis	2
Chronic granuloma	1
Abscess	1
Pneumococcal pneumonia	1

or have not had bronchograms, and the confirmation of the disease was assumed on the general appearance of the lesions.

The duration of symptoms in these 32 patients was from five days to 22 years. In the bronchiectatic group, cough, fatigue, and sputum production were the most common symptoms found. Dyspnea and chest pain followed in order, and hemoptysis was only encountered in three of 14 cases. In the carcinoma group, cough, fatigue, weight loss, and pain were the most common findings.

In all of these cases of lingular disease, cough has been a predominating symptom. The production of sputum has varied anywhere from none to one-half cupful per day. We feel that if the sputum is above this amount there is probably more than the lingular segment involved. Hemoptysis occurred in 11 cases; it was severe in only four, and two of these were bronchiectatic cases. The patient who had a granuloma of the lung had a severe hemoptysis. Three of the four cases of carcinoma had hemoptyses from slight to severe.

Dyspnea is an interesting symptom. It can be well explained in those patients who have a considerable degree of atelectasis associated with pulmonary disease. However, in this group of patients who had only one apparent segment involved, it is hard to explain.

It is important to note that several of the above cases had unexplained fever of long duration. Their x-ray films had been interpreted as normal, but when reviewed had the typical lingula sign. Further study and operation confirmed the presence of suppurative lingulitis.

Chest pain was present in 18 of the 32 cases. This was essentially of three types: (1) pleuritic, over the anterior chest in the region of the lingular segment; quite often there is associated chest wall tenderness with this type of pain; (2) pleuritic pain referred to the lateral chest wall through the lateral cutaneous branch of the intercostal nerve involved; (3) deep chest pain; this was thought to be caused by the inflammatory changes in the lingular segment or adhesions to the pericardium. A few patients had pain referred along the course of the phrenic nerve to the left shoulder.

Fatigue was a common symptom in this group of patients, and it was probably on the basis of long-standing pulmonary infection.

Fourteen of the 32 patients have been subjected to exploration; six had bronchiectasis, and two others with bronchiectasis await surgery. All of the carcinomas have been explored, and pneumonectomy was done in three cases. In the remaining resected group were patients with chronic interstitial pneumonia, lung abscess, and chronic granuloma.

The operative technique for lingulectomy used by us was essentially that described originally by Churchill and Belsey[1] and later by Overholt.[2]

There was no death in this series. One patient had a small leak in his bronchus for a time and developed a small area of abscess in the region of the resected lingular segment, but this resolved by bronchoscopy and chemotherapy, and he has been in fair condition since. The follow-up on these cases shows that all the patients having had pneumonectomy are living from five to 18 months following resection. All patients with bronchiectasis are asymptomatic at this time. The results in those cases with chronic interstitial pneumonitis have been only fair; they continue to bring up some sputum and to have cough and pain. The lung abscess and the granuloma are asymptomatic at this time.

Bronchoscopy was done on all cases with chronic lingular disease. On bronchoscopy one can find evidence of an inflammatory lesion in the region of the lingular segment, and in the cases of carcinoma positive biopsy was obtained in three of the four instances. Pus is often seen coming from the lingular bronchus, and by the use of the fore-oblique or right angle lens, one may see into the orifice of the lingular bronchus. In every case an attempt was made to pass a probe down this bronchus in order to determine if obstruction exists. Following bronchoscopy, bronchography was done if indicated, and it is important to emphasize that the absence of filling of the lingular segment should not rule out or in the diagnosis of lingular disease. Repeated bronchograms may

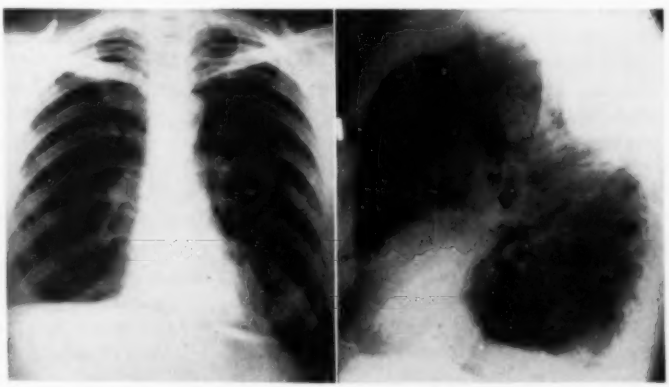


FIGURE 1a FIGURE 1b

Figure 1a: Frontal projection showing infiltrate lateral to cardiac silhouette.
Figure 1b: Lateral projection of lingular infiltrate showing triangular configuration.

be necessary before positive diagnosis can be obtained. Often the lingular segment may be filled with thick secretions and the penetration of the contrast media may be stopped before it can fill the segments.

All patients have been treated for their infection preliminary to surgery, and in some cases, intensive chemotherapy, combined with bronchoscopy and bronchography, has relieved symptoms to a considerable degree. We feel, however, that if the lingular segment is destroyed and if the patient's symptomatology warrants it, resection should be carried out.

Roentgenological Appearance

In the frontal projection, the normal lingula is roughly triangular in shape, with a transverse superior border at the hilar level, a medial border against the heart, and an oblique lateral border extending downward and medially toward the diaphragm (Figure 1a). In the lateral projection, the shadow is again triangular in shape, with apex at the hilus and base on the diaphragm; it is largely superimposed on the heart shadow (Figure 1b). In disease, its inferior extent is exceedingly variable, due to alterations in its volume.

Lesions within the lingula are best studied with the combination of a routine frontal and lateral view. For further information, bronchograms are needed in certain types of lesions, particularly bronchiectasis. On bronchograms, the lingular bronchi are best visualized on films made in the left lateral (with iodized oil in the left side only) and right anterior oblique projections. The

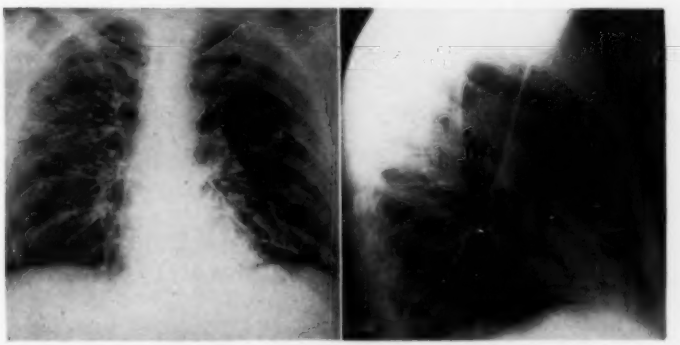


FIGURE 2a FIGURE 2b

Figure 2a: Frontal projection of bronchogram. This shows difficulty in identifying lingular segments.—*Figure 2b:* Lateral projection. Here the lingular segments are well defined.

FIGURE 3a FIGURE 3b FIGURE 3c

Figure 3a: Virus pneumonitis of lingula.—*Figure 3b:* Beginning resolution of process.—*Figure 3c:* Complete healing, showing how sharp cardiac border has returned.

lingular bronchi on a frontal view are difficult to evaluate because of the superimposed basilar divisions of the lower lobe (Figures 2a and 2b). Tomography is indicated in certain types of lesions, especially nodular and cavitating. Angiocardiography is also useful when indicated, particularly in a study of the vascular alterations accompanying neoplasms.

When the lingula is diseased, certain characteristic changes are present in most cases. Infiltrations may be of any type which are seen in other parts of the lungs, including homogeneous consolidations, linear and patchy infiltrations, nodulations, and emphysema.

One outstanding finding is almost constantly present on the frontal view, and this is often the first clue to the presence of a lesion. This finding is a partial or complete obliteration of the normally sharp left cardiac border. The distortion is primarily due to pleuro-pericardial adhesions. These adhesions between the visceral pleura and the parietal pericardium have been noted in all but one of the surgically resected cases of bronchiectasis. On the one case without adhesions, the frontal film showed a smooth cardiac border.

Poor definition without distortion of the cardiac border is most commonly seen in acute lesions such as virus pneumonia, and with complete healing, there is a return of the sharp definition of the border (Figure 3).

In acute diseases of the lingula, the segment usually is of normal volume. However, in chronic disease, this segment is subject to considerable variation in size. Normally the lingula extends to the diaphragm inferiorly, but with processes such as bronchiectasis and interstitial pneumonitis, in which atelectasis and fibrosis usually occur, contraction results and there is shrinkage toward the left hilus. The volumetric contraction determines the level of the pleuro-pericardial adhesions, and the length of involvement roughly determines the lingular size. The lingular volume was increased in both of our cases of carcinoma of the lingular orifice. In one instance, this was due to infarction of the lingula beyond the lesion, and in the other, to an extension of the malignant growth.

When obstructive emphysema is associated with lingular disease processes, there may be areas of increased radiolucency along the left cardiac border in frontal films.

On bronchographic exploration of the lingula, particularly for bronchiectasis, there is considerable variation in the degree of filling. The iodized oil may outline the entire extent of the bronchi. On the other hand, there may be filling of only half or a third of the extent (Figure 2b), and in some cases, none at all. We

FIGURE 4

Figure 4: Lateral projection of bronchogram. Lingular bronchiectasis. Note contraction of lingular segments.—*Figures 5a and 5b:* Carcinoma of lingula.

FIGURE 5a

FIGURE 5b

believe that the failure of lingular filling is as important as good filling, provided the failure is not due to technical fault. Failure to fill is usually due to obstruction by thick tenacious secretions, stricture, or neoplasm. Sometimes a diseased lingula will be well outlined on the lateral view by arborization of the oil into the small bronchioles and alveoli of the anterior division of the left upper lobe, and the medial and anterior basilar division of the lower lobe.

SUMMARY

Of the 32 cases of lingular disease presented, 14 have been resected. The predominating disease was bronchiectasis. Carcinoma was encountered in four cases. Various other types of diseases have been seen in the lingular segment. If the patient's condition warrants, we feel that resection is the treatment of choice in lingular bronchiectasis.

It is important to realize that lingular disease can be diagnosed on plain roentgenograms of the chest. On the posterio-anterior and lateral chest films one can suspect disease, and confirmation is possible by bronchoscopy, bronchography, or exploration. Lingular disease is present more often than is usually suspected. All cases should be given careful diagnostic study, as carcinoma occurred in approximately 12 per cent of those in this series.

RESUMEN

Se han resecado 14 casos de enfermedad de la lingula entre 34 pacientes. La afección predominante fué la bronquiectasis. El carcinoma se encontró en cuatro casos. Otros tipos varios de enfermedades se han encontrado en el segmento lingular.

Si el estado del enfermo lo permite creemos que la resección es el tratamiento de elección en la bronquiectasis lingular.

Es importante darse cuenta de que la enfermedad lingular puede diagnosticarse en radiografías comunes del tórax. En la postero-anterior y en la lateral se puede sospechar la afección y la confirmación es posible por broncoscopía, broncografía o exploración quirúrgica. La afección lingular se encuentra mas a menudo de lo que se sospecha. A todos los casos debe hacerse cuidadoso estudio diagnóstico puesto que el carcinoma ha ocurrido en el 12 por ciento de esta serie.

RESUME

Sur 32 cas d'affections atteignant la lingula, 14 ont donné lieu à une exérèse. L'affection prédominante a été la dilatation bronchique. Dans quatre cas, il s'agissait de cancer. Différents autres types d'affections ont été notés dans le segment de la lingula. Les

auteurs pensent que quand l'état du malade le permet, l'exérèse est le traitement de choix de la dilatation des bronches localisée à la lingula.

Il est important de concevoir que les affections de la lingula peuvent être diagnostiquées sur une simple radiographie du thorax. Sur les clichés antéro-postérieurs et latéraux, on peut suspecter l'affection qui peut être confirmée par bronchoscopie, bronchographie ou thoracotomie exploratrice. L'atteinte de la lingula est plus souvent en cause qu'on ne croit généralement. Chaque cas doit être étudié avec le plus grand soin puisque dans 12% approximativement de la série rapportée, il s'agissait de cancer.

REFERENCES

1 Churchill, E. D. and Belsey, Ronald: "Segmental Pneumonectomy in Bronchiectasis; The Lingular Segment of the Left Upper Lobe," *Ann. of Surg.*, 109:481, 1939.
2 Overholt, R. H. and Langer, L.: "The Technique of Pulmonary Resection," *Charles C. Thomas*, Springfield, Illinois, 1949.
3 Blades, Brian and Kent, Edward M.: "Individual Ligation Technique for Lower Lobe Lobectomy," *J. Thoracic Surg.*, 10:84, 1940.
4 Schmidt, H. W.: "Bronchographic Study of 555 Cases with Special Reference to Bronchiectasis," *Ann. Otology, Rhinology, and Laryngology*, 56:793, 1947.
5 Overholt, R. H., Betts, R. H. and Woods, F. M.: "Multiple Segmental Resection in the Treatment of Bronchiectasis," *Dis. Chest*, 13:583, 1947.
6 Clagett, O. T. and Deterling, B. A., Jr.: "A Technique for Segmental Pulmonary Resection with Particular Reference to Lingulectomy," *J. Thoracic Surg.*, 15:227, 1946.
7 Ewart, William: "The Bronchi and Pulmonary Blood Vessels: Their Anatomy and Nomenclature, with a Criticism of Professor Aeby's Views on the Bronchial Tree of Mammalia and of Man," London, 1889, *J. and A. Churchill, Ltd.*, 233 pp., 20 figs.
8 Boyden, E. A. and Hartmann, J. F.: "An Analysis of Variation in the Bronchopulmonary Segments of the Left Upper Lobe of Fifty Lungs," *Am. J. Anatomy*, 79:321, 1946.
9 Jackson, C. L. and Huber, J. F.: "Correlated Applied Anatomy of the Bronchial Tree and Lungs with a System of Nomenclature," *Dis. Chest*, 9:319, 1943.
10 Foster-Carter, A. F. and Hoyle, C.: "The Segments of the Lungs," *Dis. Chest*, 11:511, 1945.
11 Boyden, Edward A.: "The Intrahilar and Related Segmental Anatomy of the Lung," *Surg.*, 18:706, 1945.
12 Robbins, L. L. and Hale, C. H.: "Roentgen Appearance of Lobar and Segmental Collapse of Lung; Collapse of Right Middle Lobe," *Radiology*, 45:260, 1945.
13 Temple, H. L. and Evans, J. A.: "The Bronchopulmonary Segments," *Am. J. Roent. and Rad. Therapy*, 63:26, 1949.
14 Brock, R. C.: "The Anatomy of the Bronchial Tree," *Oxford University Press*, London, New York, Toronto, 1947.

Myasthenia Gravis, Diabetes Mellitus and Intraventricular Block, as Co-existent Complications of Pulmonary Tuberculosis

EDWARD T. SCHANTZ, M.D.*
San Antonio, Texas

While it is recognized that there is limited value in individual case reports, it was felt that the uniqueness of the co-existent disease in the case to be described justified the presentation which follows. We are reporting a case in which myasthenia gravis and diabetes mellitus are co-existent complications of pulmonary tuberculosis which apparently is the first case of its kind to be described in medical literature.

However, after an extensive review of periodicals and standard text books, we have been able to find one report only on the co-existence of myasthenia gravis and pulmonary tuberculosis[1]

Despite the extreme frequency with which diabetes mellitus is encountered today there has been only one case reported of the co-existence of diabetes mellitus and myasthenia gravis. This accurate account was written recently by Perry[2] and he states that his is the first recorded case of the association of these two diseases.

Of course, no attempt is being made to connect the etiological factor of myasthenia gravis and pulmonary tuberculosis—the co-existence being purely coincidental.

Case Report: D.D. is a 48-year-old white male whose pulmonary symptoms dated to August of 1949 at which time he had several bouts of hemoptysis. Pulmonary tuberculosis was diagnosed and he quit his job and went to bed at home. He continued at bed rest and had no other form of therapy. He was admitted to the Hamburg State Sanatorium in June of 1950 for further treatment.

Past Medical History: The patient has been a known diabetic for the past 12 years requiring 40 units of protamine zinc insulin daily. He states that he had one bout of diabetic coma in 1943. In 1944 he suddenly developed drooping of the eyelids associated with generalized weakness and was admitted to a hospital where a diagnosis of myasthenia gravis was made. Shortly thereafter he became unable to swallow, and the weakness became progressively worse. He received several injections of prostigmine with dramatic recovery. With subsequent injections the ptosis of the eyelids (especially the left) failed to respond to prostigmine. However the difficulty in swallowing and the generalized weakness practically disappeared.

*Formerly Resident, Pulmonary Diseases, Pennsylvania State Sanatorium for Tuberculosis, No. 3, Hamburg, Pennsylvania.

Physical Examination: On admission we noted a fairly well-developed and well-nourished white male in no acute distress. His temperature was 98.8 degrees F., pulse 78, and respiration 18. Ptosis of both eyelids (Figure 1), more marked on the left, was present but there did not seem to be any disturbance in the extra-ocular movements. His pupils were equal and reacted to light and accommodation. Ear, nose, and throat examination was essentially negative.

Examination of the heart revealed no audible murmurs or palpable thrills, however, just to the left of the sternum, reduplication of the first heart sound could be heard. The apex rate was 80 per minute, blood pressure: right 162/104, and left 154/100.

Physical signs were absent over the lungs except for some coarse rhonchi over the left upper lobe posteriorly.

Neurologically, aside from the ptosis of the lids, nothing remarkable could be noted. He had moderately good motor strength in both the arms and the legs. Deep tendon reflexes were present and were bilaterally equal. No pathological reflexes could be noted.

Impressions on admission were: (1) Pulmonary tuberculosis. (2) Diabetes mellitus. (3) Myasthenia gravis.

Laboratory Examinations were irrelevant, except 2 plus sugar in the urine and blood sugar, 360 mg. per cent.

Following admission an extensive course of sputum concentrates were negative, but the tubercle bacillus was finally obtained in a culture of

FIGURE 1: Patient D.D. Ptosis of both lids was present on admission, being more marked on the left.

the gastric washings. Histoplasmin and coccidioidin skin tests resulted in no reaction.

Roentgenogram of the chest revealed evidence of infiltration in both upper lobes with a definite cavity in the left upper lobe (Figure 2a).

Fluoroscopic Findings: The cardiac silhouette seemed to be slightly larger than normal with the apex pointing downward. No abnormality could be seen on the right border but on the left side the aortic knob was quite prominent. In the right oblique view, the barium meal showed no definite abnormality. In the left oblique view, the left ventricle seemed enlarged. Both diaphragmatic shadows were clear and there was good function of the diaphragms. Bronchoscopic examination failed to reveal any endobronchial pathology.

Electrocardiographic Findings: Rate, 90 per minute; Mechanism, Intraventricular block; Axis, Normal; PR Interval, 0.20 seconds; QRS Interval, 0.12 seconds. Broad S waves are noted in the standard limb leads and the unipolar pre-cordial leads; ST Segments, normal; T Waves, normal.

Progress While in the Hospital: In view of the cavitary lesion in the

FIGURE 2a

FIGURE 2b

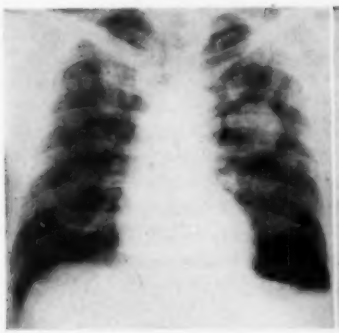


FIGURE 2c

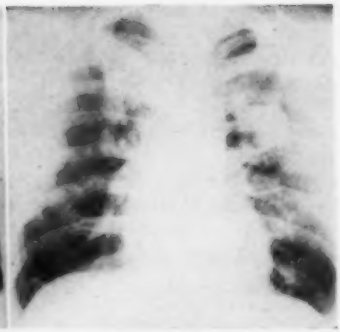


FIGURE 2d

Figure 2: (a) Admission plate revealing cavity in left upper lobe.—(b), (c), and (d) Subsequent plates showing selective collapse of left upper lobe with gradual closure of cavity.

left upper lobe, it was deemed advisable to institute left artificial pneumothorax. This was started successfully with a selective collapse of the left upper lobe despite some adhesions (Figure 2b). The latest x-ray films show apparent closure of the cavity (Figures 2c and 2d). Artificial pneumothorax is being continued at present with no complications.

The patient's clinical condition has remained excellent. He has gained five pounds of weight since admission and his diabetes has been adequately controlled on 40 to 50 units of protamine zinc insulin daily.

Although ptosis of the lids persists and is somewhat annoying at times, he has shown no tendency to develop a relapse of the myasthenia gravis, and further prostigmine therapy has not as yet been necessary.

In addition to the three diseases described above, it is interesting to note that the patient exhibited an intra-ventricular block on the electrocardiogram. Baark[3] in 1945, described a case of bundle branch block in a patient with myasthenia gravis. He concluded that these two conditions were independent, and the cardiac findings were most likely due to coronary artery disease. As it is not uncommon for diabetics to develop coronary heart disease at an early date, the electrocardiographic findings noted above are likewise probably not related to the myasthenia gravis but represent coronary sclerosis.

SUMMARY

1) A case of pulmonary tuberculosis complicated by myasthenia gravis and diabetes mellitus has been described. It is felt that this is the first case of its type reported in medical literature.

2) There probably is no relation between pulmonary tuberculosis and myasthenia gravis.

RESUMEN

1) Se relata un caso de tuberculosis pulmonar complicado con mistenia grave y diabetes. Se cree que este es el primer caso descrito en la literatura.

2) Probablemente no hay relación entre la tuberculosis y la miastenia grave.

RESUME

1) L'auteur décrit un cas de tuberculose pulmonaire compliqué de myasthénie et de diabète sucré. Il semble qu'il s'agisse du premier cas de ce type rapporté dans la littérature médicale.

2) Il n'y a probablement aucune relation entre la tuberculose pulmonaire et la myasthénie.

REFERENCES

1 Rothstein, E. and Pirkle, H. B.: "Myasthenia Gravis," *Am. Rev. Tuberc.*, 49:381, 1944.
2 Perry, S.: "Diabetes Mellitus in Association with Myasthenia Gravis," *J.A.M.A.*, 143:1332, 1950.
3 Baark, V.: "Intermittent Bundle Branch Block in a Patient with Myasthenia Gravis," *Acta. Med. Scandinavian*, 121:295, 1945.

Basal Metabolism in Pulmonary Tuberculosis

Patients Under Collapse Therapy*

GEORGE C. LEINER, M.D. and SOL ABRAMOWITZ†
Staten Island, New York

On the basis of 567 determinations of the basal metabolic rate performed on 394 patients with pulmonary tuberculosis it was found in a previous publication[1] that there was a tendency to a low basal metabolic rate in these patients. Furthermore it was noted that in tuberculous patients the basal metabolic rate usually showed a tendency to decrease when they were on bed rest and under collapse therapy.

Numerous studies of the basal metabolic rate done by us since then have given similar results. Among the patients in whom these examinations were done, were many who had undergone various forms of collapse procedures.

Epstein and Pine[2] did determinations of the basal metabolic rate in patients with unilateral pneumothorax, with bilateral pneumothorax and with unilateral thoracoplasty and found a lowered rate in a good percentage of this group.

Topper and Rubin[3] also found that in children there was a decrease of the basal metabolic rate following pneumothorax induction.

Since these basal metabolic rate studies were done in a relatively small number of patients and since they were done only in some forms of collapse therapy we thought it would be of interest to summarize the results we have obtained in a large number of tuberculous patients under various forms of collapse therapy.

Material and Method

The method was the same as outlined in a previous paper.[1] The age of the patients ranged between 16 and 60 years. Most of them had far advanced pulmonary tuberculosis, in a few only the disease was moderately advanced. All were afebrile at the time of their examination.

In the 11 years between 1939 and 1950, 185 determinations of

*From the Pulmonary Physiology Laboratory, Halloran Veterans Administration Hospital, Staten Island, New York, and Division of Pulmonary Diseases, Montefiore Hospital, New York, New York.

†Sponsored by Veterans Administration and published with approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

the basal metabolic rate were done on 145 patients with pulmonary tuberculosis who were under some form of collapse therapy. The examinations were done when the collapse was well established: In patients with pneumoperitoneum several weeks after induction of the pneumoperitoneum, in patients with pneumothorax, thoracoplasty or phrenic nerve crush, several months after the collapse had been achieved. When more than one examination was done on the same patient the average of the findings was taken.

Results

1) *Pneumothorax:* There were 55 patients with artificial pneumothorax (Figure 1). Twenty-seven (49.2 per cent) had a normal basal metabolic rate (between −10 per cent and +10 per cent). A diminished rate (< −10 per cent) was found in 25 patients (45.4 per cent). It was found increased (> +10 per cent) in three patients (5.4 per cent).

Further analysis of these figures reveals that a basal metabolic rate between −11 per cent and −20 per cent was found in 23 patients (41.8 per cent), and between −20 per cent and −30 per cent in two (3.6 per cent).

In the patients with pneumothorax the basal metabolic rate ranged between −24 per cent and +17 per cent. The average was −7 per cent, with a standard deviation of 9.2.

There were seven with bilateral pneumothorax. The average rate in these was −7 per cent with a standard deviation of 8.8.

2) *Pneumoperitoneum:* There were 17 patients with pneumoperitoneum (Figure 2). Thirteen (76.6 per cent) had a normal basal metabolic rate (between −10 per cent and +10 per cent).

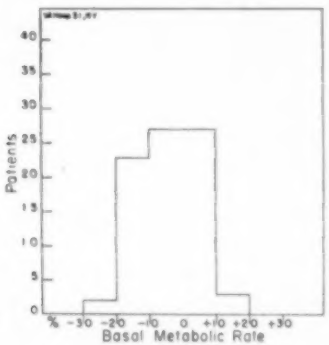


FIGURE 1

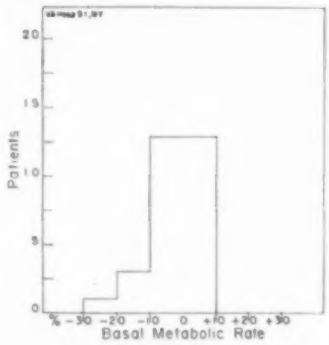


FIGURE 2

Figure 1: Distribution of basal metabolic rates in 55 patients with pneumothorax.—*Figure 2:* Distribution of basal metabolic rates in 17 patients with pneumoperitoneum.

Four (23.4 per cent) had a diminished rate (< -10 per cent). Of these, three had rates between -11 per cent and -20 per cent, and one, between -21 per cent and -30 per cent. In none was an increased rate found.

In the pneumoperitoneum group the basal metabolic rate ranged between -21 per cent and $+10$ per cent. The average was -6 per cent, with a standard deviation of 7.7.

3) *Thoracoplasty:* There were 65 patients who had undergone thoracoplasties of various extent (4 to 8 ribs) (Figure 3). Of these, 39 (60 per cent) had a normal basal metabolic rate (between -10 per cent and $+10$ per cent). Twenty-one (32.3 per cent) had a diminished rate (< -10 per cent). An increased rate (> -10 per cent) was found in five (7.7 per cent).

A further break-down of these observations shows that 20 (30.8 per cent) had a basal metabolic rate between -11 per cent and -20 per cent, and one patient (1.5 per cent), between -21 and -30 per cent. Four (6.2 per cent) had a rate between $+11$ per cent and $+20$ per cent, one (1.5 per cent), between $+21$ per cent and $+30$ per cent.

In the patients with thoracoplasty the basal metabolic rate ranged between -23 per cent and $+23$ per cent. The average was -5 per cent, with a standard deviation of 10.1.

In addition, there were three in whom paraffin plombage had been done. Their rate ranged between -16 per cent and $+10$ per cent. The average was -4 per cent.

4) *Phrenic crush:* There were only five patients in whom a phrenic crush had been done. One of them (20 per cent) had a

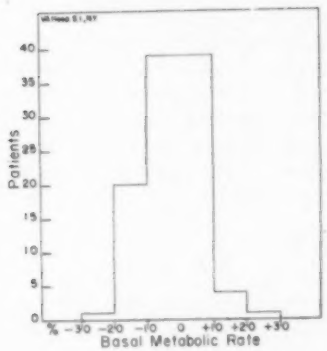


FIGURE 3

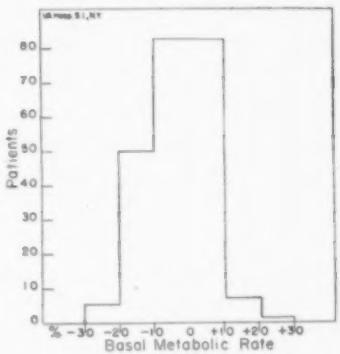


FIGURE 4

Figure 3: Distribution of basal metabolic rates in 65 patients with thoracoplasty.—*Figure 4:* Distribution of basal metabolic rates in 145 patients with various forms of collapse therapy.

TABLE I: BASAL METABOLIC RATE IN PATIENTS UNDER COLLAPSE THERAPY

Form of Collapse Therapy	No. of Patients	Range of Basal Metabolic Rate Per cent	Mean Basal Metabolic Rate Per cent	— — NORMAL — — (−10 to +10%) No.	Per cent	DECREASED (<−10%) No.	Per cent	INCREASED (>+10%) No.	Per cent
Pneumothorax	55	(−24, +17)	−7	27	49.2	25	45.4	3	5.4
Pneumoperitoneum	17	(−21, +10)	−6	13	76.6	4	23.4	0	0
Thoracoplasty	65	(−23, +23)	−5	39	60.0	21	32.3	5	7.7
Paraffin Plombage	3	(−16, +10)	−4	2	66.7	1	33.3	0	0
Phrenic nerve crush	5	(−22, −7)	−14	1	20.0	4	80.0	0	0
All patients	145	(−24, +23)	−6	82	56.6	55	37.9	8	5.5

normal basal metabolic rate. Four (80 per cent) had a diminished rate (< −10 per cent). Of these, three had rates between −11 per cent and −20 per cent, and one, between −21 per cent and −30 per cent. In no patient was an increased rate found.

In the patients with phrenic crush the basal metabolic rate ranged between −22 per cent and −7 per cent. The average was −14 per cent, with a standard deviation of 5.7.

5) *All forms of collapse therapy:* Altogether, there were 145 patients with pneumothorax, pneumoperitoneum, thoracoplasty, paraffin plombage or phrenic nerve crush in whom studies of the basal metabolic rate were done (Figure 4). Eighty-two (56.6 per cent) had a normal rate. A diminished rate was found in 55 (37.9 per cent). It was found increased in eight (5.5 per cent).

Among the patients with a decreased basal metabolic rate there were 50 (34.4 per cent) in whom it ranged between −11 per cent and −20 per cent and five (3.5 per cent), between −21 and −30 per cent. An increased rate between +11 and +20 per cent was seen in seven (4.8 per cent), between +21 and +30 per cent in one (0.7 per cent).

In all the patients with some form of collapse procedure the basal metabolic rate ranged between −24 and +23 per cent. The average was −6 per cent, with a standard deviation of 9.4.

Discussion

The findings are summarized in Table I. The results in the various groups are similar. No significant difference of the basal metabolic rate is found between patients with pneumothorax and thoracoplasty, which are the two largest groups.

In a previous study[1] it was found that 64.9 per cent of 567 examinations in patients with pulmonary tuberculosis gave a rate within normal limits. A diminished rate was found in 21.0 per cent, an increased rate in 14.1 per cent. This tendency to a slightly subnormal basal metabolic rate is even more pronounced in patients with some form of collapse therapy: 37.9 per cent of the patients had a subnormal rate and only 5.5 per cent an increased rate. These findings agree with those of other authors.[2,3]

SUMMARY

Basal metabolism studies were done in 145 patients under various forms of collapse therapy for pulmonary tuberculosis. A normal rate (between −10 per cent and +10 per cent) was found in 82 patients (56.6 per cent). The rate was subnormal in 55 patients (37.9 per cent). It was above normal in 8 patients (5.5 per cent).

Of 55 patients with artificial pneumothorax, the rate was normal

in 27 (49.2 per cent). It was diminished in 25 (45.4 per cent). It was increased in three (5.4 per cent).

Of 17 patients with pneumoperitoneum, the rate was normal in 13 (76.6 per cent). It was diminished in 4 (23.4 per cent).

Of 65 patients with thoracoplasty, the rate was normal in 39 (60.0 per cent). It was diminished in 21 (32.3 per cent). It was increased in 5 (7.7 per cent).

Of five patients with phrenic nerve crush, the basal metabolism was normal in one and diminished in four.

Patients with pulmonary tuberculosis under collapse therapy show a more pronounced tendency to a subnormal basal metabolic rate than those without collapse therapy.

RESUMEN

Se hicieron estudios del metabolismo basal en 145 enfermos de tuberculosis, bajo diversas formas de colapso.

Se encontró un metabolismo normal entre 10 por ciento y 110 por ciento, en 82 enfermos (56.6 por ciento).

En 55 enfermos (37.9 por ciento) el M.B. fué subnormal, y en 5.5 por ciento el M.B. fué arriba de lo normal.

De 55 enfermos con neumotorax artificial, el M.B. fué normal en 27 (49.2 por ciento), disminuido en 25 (45.4 por ciento) y aumentado en 3 (5.4 por ciento).

De 17 enfermos con neumoperitoneo, el M.B. fué normal en 13 (76.6 por ciento), disminuido en 4 (23.4 por ciento).

RESUME

Le métabolisme basal à été étudié chez 145 malades traités par différentes variétés de collapsothérapies pour tuberculose pulmonaire. 82 malades (56.6%) avaient un métabolisme normal (entre −10 et +10 pour cent). Les résultats furent subnormaux chez 55 malades (37.9%) et chez 8 malades (5.5%) au-dessous de la normale.

Parmi 55 malades traités par pneumothorax, les chiffres furent normaux chez 27 d'entre eux (49.2%), diminués chez 25 malades (45.4%), augmentés chez trois malades (5.4%).

Parmi 17 malades traités par pneumopéritoine, le métabolisme était normal chez 13 d'entre eux (76.6%) et diminué chez les quatre autres malades (23.4%).

REFERENCES

1 Leiner, G. C.: "Basal Metabolism in Pulmonary Tuberculosis," *Am. Rev. Tuberc.*, 50:223, 1944.

2 Epstein, H. H. and Pine, I.: "Studies on the Basal Metabolic Rate in Pulmonary Tuberculosis," *Quart. Bull. Sea View Hosp.*, 1:52, 1935.

3 Topper, Anne and Rubin, H. S.: "Basal Metabolism of Tuberculous Children: IV. Children with Pneumothorax," *Am. J. Dis. Child.*, 58: 959, 1939.

Pleuro-Pneumonectomy in the Treatment of Tuberculous Empyema

JOS DAEMS, M.D. and ROBERT PANNIER, M.D.*
Bruges, Belgium

The treatment of tuberculous empyema still remains a major therapeutic problem in tuberculosis. In recent years, the use of antibiotics and pleural lavages have undoubtedly improved the prognosis of uncomplicated tuberculous empyemata; however, when a broncho-pleural fistula with subsequent mixed infection empyema is present, major thoracic intervention will still be the only way to obtain a complete cure.

Lately this treatment consisted in draining the pleural cavity with a subsequent thoracoplasty, which most of the time had to be completed by a resection of the parietal pleura and one or more plastic operations. Even in the successful cases this long and painful surgical treatment finally leaves a greatly deformed and crippled patient.

In this short note we shall not consider the technical side of this problem on which Daems (thoracic surgeon) will report shortly. We also refer to De Winter's report on 1,137 cases of tuberculosis treated by thoracoplasty.[1] An analysis of the experience of several authors shows the high mortality rate of these multiple surgical interventions, which most of the time have to be carried out on patients in bad physical condition.

At the Hopital St. Jean in Bruges, 58 cases of broncho-pleural fistula with mixed empyema have been surgically treated with drainage, thoracoplasty and pleurectomy between January 1, 1945 and January 1, 1950. The results are as follows:

Deaths	38 (65 per cent)
Incompletely cured	5 (10 per cent)
Completely cured	15 (25 per cent)

These results are similar to those obtained by most of the authors who have reported on that subject. The great mortality rate in this operation is mainly due to postoperative shock. A few died because of toxaemia, generalized amyloidosis and of tuberculous spread to the contralateral lung.

Of the five cases incompletely cured, the failure was due in three to the persistance of a tuberculous sinus; in one to a contralateral tuberculous spread and in one to paralysis subsequent

*From the Department of Lung Diseases (Dr. L. DeWinter, Director), Hopital St. Jean, Bruges, Belgium.

to myelitis which occurred after the surgical treatment. Those who are now completely cured remained in hospital for eight to 10 months on the average.

Considering the fact that after the failure of conservative treatment, all these cases were irremediably lost, we may consider even the meagre results obtained by surgical collapse therapy as satisfactory. It remains an inadequate procedure however.

It is therefore not astonishing that after the successes of excisional surgery in pulmonary tuberculosis some surgeons have been tempted by the idea of accomplishing the removal "en bloc" of the diseased lung and the infected pleural sack. Experience in this field is not yet widespread, but the first results obtained with pleuropneumonectomy by Sarrot, Overholt, Santy, indicate that with this less mutilating procedure the chances of final healing are much higher, and can be obtained more rapidly.

Since 1948, 12 cases of infected tuberculous empyemata, of which 11 had a bronchopleural fistula were treated by pleuropneumonectomy in the Hopital St. Jean, Bruges. Moreover one patient had a parietal fistula and three had tuberculous lesions in the other lung.

All these operations have been performed under high spinal anesthesia. Immediately before operation the pleural sack is emptied under endoscopic control. The parietal pleura and the lung are dissected in the extrapleural plane to lessen the risk of cutting into the pleural sack or the diseased lung parenchyma.

It is however not always technically possible to perform the extrapleural dissection without tearing the parietal pleura. Sometimes the parietal pleura is so adherent to the parietal tissues that the surgeon is forced to tear the pleura into shreads. No post-operative empyema developed in these cases, despite the fact that the extrapleural space was contaminated during the operation. Up to now the results are as follows:

Pleuro-pneumonectomy, 12; deaths, 4: One because of post-operative gastric dilatation (case with parietal fistula). One because of contralateral spread (in spite of the induction of an artificial pneumothorax). One developed tuberculous meningitis three months after operation (case with bilateral tuberculosis). One died eight months after the operation because of a thoracoplasty performed for empyema of the extrapleural space (bronchial fistula).

Incompletely healed, 1: This patient developed a small bronchial fistula which healed spontaneously (case with contralateral disease controlled by pneumothorax).

Completely healed, 7 (between two years and six months). None developed empyema after the operation. Only two patients had

thoracoplasty performed after pleuro-pneumonectomy. The posterior thoracoplasty was performed at the same time as the pleuropulmonary resection.

This first experience with pleuro-pneumonectomy in the treatment of infected tuberculous empyemata complicated with bronchopulmonary fistula is yet too recent to be conclusive. However the results obtained up to now are very encouraging, as the number of successes have gone up from 25 per cent with surgical collapse therapy to 65 per cent with pleuro-pneumonectomy.

These figures will still improve when the patients, freed from the fear of a long, dangerous and finally mutilating operation will be brought to the surgeon as soon as the failure of a conservative treatment is stated.

It is our opinion that pleuro-pneumonectomy must now be considered as the operation of choice in the treatment of tuberculous empyemata with broncho-pleural fistula, and in cases without bronchial fistula rebellious to any medical treatment.

The sufferings and the misery of surgical collapse therapy must only be reserved to the cases where removal of the diseased lung with the infected caseous parietal pleura is technically impossible.

SUMMARY

A comparative study of the results obtained with surgical collapse therapy and with pleuro-pneumonectomy in the treatment of infected tuberculous empyemata with or without bronchopleural fistula, shows that the latter method must now be advocated as the most adequate treatment for these tuberculous conditions.

RESUMEN

Un estudio comparativo de los resultados obtenidos con el colapso quirúrgico y con la pleuro-neumonectomía en el tratamiento de los empiemas tuberculosos con o sin fístula bronquial muestra que el segundo método debe ahora ser el recomendado para el tratamiento mas adecuado de estas afecciones tuberculosas.

RESUME

Les auteurs comparent le résultat obtenu par la collapsothérapie chirurgicale et par la pleuro-pneumonectomie dans le traitement des pleurésies purulentes tuberculeuses avec ou sans fistule bronchique. Cette étude montre que la pleuro-pneumonectomie est actuellement le traitement qui convient le mieux à cette forme de tuberculose.

REFERENCE

1 DeWinter, L.: "1,137 cas de tuberculose pulmonaire traités par thoracoplastie," *Acta Tuberculosea Belgica* nr. spec. 2 ieme partie 1949.

Cough: Its Pathology, Therapy and Management*

A. WORTH HOBBY, M.D., F.C.C.P.
Atlanta, Georgia

Man's most common symptom is cough. Because of its frequency, cough is often taken for granted by the patient and is overlooked by the physician. Cough is defined as a sudden violent expulsion of air after deep inspiration and closure of the glottis. The results of a cough may be either harmful or helpful to the patient. The cause of a cough must be known to the physician if he is to appraise the results. To this end a careful and detailed history must be obtained.

The diagnosis of bronchiectasis is apparent at once when the following history is elicited: Cough, productive of one to three teacupsful of sputum per 24 hours which may or may not be blood streaked, and is not excessively offensive in odor. This cough has persisted over a long period of time and usually originates after an infection such as influenza, pneumonia, or measles. The patient is not acutely ill and has lost little or no weight.

Again the diagnosis is easily and quickly made by a history of persistent coughing with loss of weight, night sweats, a history of pleurisy and contact with active pulmonary tuberculosis. The patient may have had blood streaked sputum, hemoptysis, chest pains, afternoon elevation of temperature, loss of energy and abnormal fatigue.

All diagnoses are not so easily determined, and an intimate knowledge of the co-existing physiological conditions relating to the causes must be obtained. For example the following differences between harmful and helpful coughing must be well understood:

(1) Harmful coughing is produced by:[1-3]

- (A) Stimulation of the cough reflex by extra-pulmonary irritation due to:
  - (a) Drainage from the paranasal sinuses
  - (b) An elongated uvula
  - (c) Pressure on the trachea or bronchi by mediastinal inflammation, tumors, or dilated blood vessels
- (B) Non-inflammatory lesions of the bronchial tree such as benign or malignant tumors

*Read at the American Medical Association's Third Annual Clinical Session, Washington, D. C., December 8, 1949.

(C) Inflammatory processes of the bronchi or the lung parenchyma without the formation of exudate, but with constant irritation of the cough centers; as, in the dry cough of early acute bronchitis
(D) Mucopurulent or mucoid secretions resulting from inflammation of the bronchial tree that are too tenacious to be removed by cough, as in the paroxysmal stage of whooping cough
(E) Transudation in the alveoli during heart failure
(F) Obstruction of the bronchial tree due to pathological changes which cause retention of the secretions and exudates distal to the obstruction
(G) Atelectasis, because the definition of cough cannot be fulfilled
(H) Emphysema as result of:
  (a) Weakening and destruction of the elastic structures
  (b) Reduction of the intrapleural sub-atmospheric pressure
  (c) Abnormally low position of the diaphragm
(I) Fatigue and exhaustion of the expiratory muscles; as in fibrotic tuberculosis and pulmonary fibrosis
(J) Psychogenic cough such as that produced by the exhibitionist
(K) Allergies (Approximately 1,000,000 people in the United States have asthma)

(2) An unsuccessful or harmful cough is found especially in pulmonary tuberculosis and may result also in:

(A) Pulmonary hemorrhage
(B) Laryngitis
(C) Spontaneous pneumothorax
(D) Vomiting
(E) Loss of appetite
(F) Exhaustion
(G) Headache
(H) Insomnia
(I) Elevated temperature
(J) Marked dyspnea
(K) Cyanosis
(L) Thoracic pain
(M) Fracture of ribs
(N) Mediastinal emphysema
(O) Subcutaneous emphysema
(P) Subconjunctival hemorrhage
(Q) Urinary incontinence
(R) Myocardial failure

In contrast a helpful cough has as its purpose the removal of:[2] abnormal mucous, inflammatory products, foreign bodies, products resulting from circulatory congestion and all irritating sensations in the respiratory tree. To be successful, a cough must produce effective drainage of the lung during an inflammatory disease without undue stress on any of the tissues of the body.

The need for successful management of coughing is emphasized by the possibility of contagion in infectious diseases. It is estimated that an average of two and one-half colds occur for every man, woman and child in the United States each year. A person talking in a normal conversational tone sprays out sputum for a distance of eight feet. A cough carries sputum 12 to 15 feet, and a sneeze may propel particles as much as 20 feet. An analysis[4] of airborne transmissions of disease has shown that tubercle bacilli may travel by droplets no larger than 0.2 mm. which quickly clear from the air by gravity. Droplet nuclei, less than 0.1 mm., which quickly evaporate, continue to float and are a dangerous cause of infection.

"The cough center is located in the medulla near the sensory portion of the vagus nerve and also near the vomiting center. The cough center is subject to both peripheral and central stimuli and is depressed by drugs such as the alkaloids of opium, alcohol and anaesthetic agents. Coughing is provoked by the stimulation of the sensory endings of the glossopharyngeal and vagus nerves."[1-3] Eight groups of branches of the vagus nerve are associated with cough. They are described as: Arnold's branch to the ear which is stimulated by wax, eczema, etc., causing a cough; the pharyngeal branch; the superior laryngeal branch sensory to the base of the tongue and larynx and is frequently a cause of coughing. Among this group is also found the inferior laryngeal branch (motor), which may produce an inefficient cough; and cardiac branches, which are indirect causes through circulatory failure. The last three in this group are listed as: the pulmonary branches which are concerned in the cough of gross pulmonary or pleural diseases; the esophageal and precardial branches; and the gastric branches which occasionally cause coughing due to dyspepsia.

The irritants to the surfaces enervated by the distribution of these nerves are: foreign bodies as dust, food, tobacco, smoke, etc.; excess natural secretions; pressure and inflammation; and acute or chronic simple debility or increased irritability. The secretory function of the glands of the bronchial mucosa is increased by stimulation of these vagal nerve endings.

Rhythmic relaxation and contraction of smooth muscles in the wall of the bronchioles is a normal process. Occlusion of the smaller branches of the bronchial tree is caused by spasm of these muscles. This may result from allergies and irritants; as, gases, fumes, and foreign bodies, and may lead to dyspnea, anoxemia, atelectasis, and increased cough.

Bronchi and bronchioles have a peristaltic motion directed from the smaller structures toward the larger structures. It is inde-

pendent of respiratory movement. Drugs of the parasympathomimetic group, which cause bronchospasm, also decrease or abolish bronchial peristalsis. Inhalation of carbon dioxide and oxygen in the proper mixture increases this peristaltic motion.[1-3]

Cilia in larger and smaller bronchi normally drive inert material toward the larynx. Destruction of the sensory nerve endings of the vagus (by any pathological process) destroys the ciliary function. Absence of the cilia; as, in bronchiectasis, deprives the lung of this much needed help. Alkaline solutions stimulate ciliary motion. The normal motion of bronchial secretions toward the larynx may be unfavorably influenced by drugs that change their consistency or amount such as morphine which suppresses normal bronchial secretions.

The pharmacology of cough[5] control may be divided into: local applications; medication transported through the blood stream to the mucous membrane of the respiratory passages; and medication which stimulates the central nervous system. Local applications may be soothing to the mucous membrane as lozenges or hyperemic when produced by inhalations of vapors of volatile oils such as eucalyptol and creosote. A third group of local medications include aerosols of a bacteriastatic nature combined with mucuous membrane strinking agents. The most common blood borne drugs used for aiding the cough mechanism are ammonium chloride, potassium iodide, and creosote. Direct action on the central nervous system is demonstrated by carbon dioxide inhalations. Paregoric acts indirectly by irritating the vagus nerve endings in the gastric mucosa.

Now that the cause of cough has been determined and its physiology is understood, the treatment is to be considered. When tumor, foreign body, elongated uvula, dilated blood vessels, and atelectasis are found to be the cause, appropriate surgery is most often indicated. Surgery is needed often in infections; such as, bronchiectasis, lung abscess, empyema, and other pulmonary complications. Deep x-ray therapy may or may not be indicated, alone or in conjunction with surgery in the above conditions. The presence of infection, the type of infection, the location, extent and character of the lesion will indicate the treatment. It goes without saying that the cure of the disease stops the cough. The treatment of the various diseases of which cough is a symptom is beyond the scope of this paper.

The amount of secretion expectorated is not a true index because some of the secretion is resorbed, especially where it is thick or scanty. Resorption is done by a process of digestion or dilution and liquifaction through the admixture of the secretions of the glands of the bronchial mucosa and the alveoli.[6] The re-

sorptive capacity of the lungs can be effectively increased by oral administration of ammonium chloride, potassium iodide, fluid extract of senega, fluid extract of ipecac, emetine hydrochloride, and by inhalation of oxygen under positive pressure.

When indicated, drug therapy should be applied judiciously. The Manual of Pharmacology, 1948, seventh edition by Sollmann and the Pharmacological Basis and Therapeutics, 1941, by Goodman and Gilman, enumerate and classify expectorants under the heading of anodyne, sedative, and stimulative expectorants.

(1) Anodyne expectorants depress the cough reflex by central action.

(A) Morphine alkaloids depress the irritative reflex and decrease the amount of bronchial secretions. They are used when the cough is dry and hacking, but are contraindicated when excessive mucous is present. Codeine, tincture of opium, and pantopon are the most commonly used. Heroin should be avoided, because of its habit forming tendency.

(B) Piperidine (sedulon) depresses the medullary centers, especially the cough center.

(2) Sedative expectorants tend to alleviate the inflammatory process by promoting the secretion of protective mucous, thus decreasing the frequency of paroxysms of cough. They are divided into three groups, each acting in a different way:

(A) Saline expectorants, although their exact mode of action is not known, are believed to act reflexly, stimulating bronchial secretions through local gastric irritation. They are: ammonium chloride, for "tight" coughs; ammonium carbonate, for liquifying thick, purulent mucous; iodides, for increasing bronchial secretions by rendering them less viscid. They are used preferably in chronic cases to "loosen" the cough. Citrates and acetates of sodium are used as expectorant drugs primarily on an emperical basis.

(B) Nauseant expectorants increase the secretion and lessen the viscosity of bronchial mucous. In addition to their expectorant effect, they have a nauseant and emetic action which is chiefly central in origin. Such drugs are ipecac, emetine, tartar emetic, apomorphine, salts of ammonium (especially the chlorides and carbonates), and drugs acting through the saponins, which include squill, senega, and grindelia.

(C) Demulcent expectorants diminish the tendency to cough by a local soothing effect on the inflamed mucosa of the

pharynx. Such drugs are: syrup of acacia, licorice, glycerin, cane sugar, "cough drops," and hot drinks.

(3) Stimulant expectorants are mildly irritating in nature, but most of them will diminish bronchial secretion. Among these drugs are: creosotes (calcium creosotate and creosote carbonate), guaiacol (potassium orthoguaicol-sulfonate in a syrup), terpin hydrate, balsams of Tolu and Peru, tar, and terpin. To this may be added the drugs described by Young:[7]

(A) Antispasmodic drugs (broncho-dilator or antispasmodic), which include atropine, belladonna, stramonium, lobelia, hyoscyamus, epinephrine, ephedrine, nicotine, and papaverine.

(B) Other drugs that may be used to promote expectoration are: strychnine, coramine, and cardiazol. Other agents such as: liniments, poultices, heat, diathermy, and oxygen also may be employed.

The role of respiratory motion in the removal of inflammatory products from the bronchi is important. This rhythmically repeated motion tends mechanically to remove secretions.

Certain gases such as carbon dioxide and oxygen aid in increasing respiratory motion and, in turn, the removal of inflammatory products from the bronchi. Shallow breathing is harmful and is usually found in debilitated patients, or as an effect of general anesthesia, or in depression of the respiratory center by narcotics, or in anoxemia which may excite more vigorous breathing[8] only to be followed by greater depression of respiration.

Secretions in the smaller bronchi do not produce effective cough. They are only effective in the larger bronchi; therefore, a pathological process localized in the bronchioles, such as, in bronchiolitis, may cause respiratory distress, anoxemia, and cyanosis. In the absence of an effective cough, therapeutic intervention is necessary to drain the small bronchi, but such therapy must not increase the accumulation of exudate.[1-3]

Short inhalations of 5 to 10 per cent carbon dioxide with oxygen are found to give the best results. There is an increase in the rate of respiration and in the volume of air in the lungs; the ventricular output of the heart is augmented; the systolic and diastolic blood pressures are raised; pulse rate is accelerated; and pulse pressure is elevated.

Carbon dioxide and oxygen are contraindicated "when there has been recent pulmonary hemorrhage; when the patient has marked emphysema; or wide-spread pulmonary fibrosis without atelectasis, bronchiectasis, or mucopurulent retention in the air pass-

ages. Acute plastic pleurisy, pleurisy with effusion, and hypertension, as well as cough arising from causes outside the lungs also are contraindications to the use of these gases."[1-3]

The use of antibiotics in aerosol for the treatment of pulmonary infection has been discussed extensively in the literature[9] and needs no repetition here, except to recommend that one-half cc. of epinephrine be added to each portion of aerosol (certain conditions excepted), and that large doses of the antibiotic be given intramuscularly in a vehicle that prolongs the maintenance of high levels of concentration of the drug in the blood. One aerosol treatment and one intramuscular injection per day usually is sufficient. It must be remembered that some patients respond only to double or even triple the usual dose of each intramuscular injection.

Effective concentrations of drugs given by aerosol can be demonstrated by the following[10] examples. The volume of air inspired by the lungs is approximately doubled by 1 per cent atropine (from 450 to 1000 cc.), by 5 per cent sodium nitrate (from 500 to 900 cc.), and by 1 per cent procaine hydrochloride (from 650 to 1000 cc.).

Instillations of oil, iodinized oil, and oil combined with the sulfonamides or the antibiotics may be used effectively in a small number of instances. It has been argued that instillation of oil renders the cilia useless, and therefore, should not be used. However, from a practical standpoint, in such diseases as bronchiectasis, temporary improvement in the cough and other symptoms is seen occasionally. The patient should be told that this method is a temporary measure and that the underlying disease will have to be cured by other means, for unquestionably the patient is greatly impressed by its use. Oil instillations should be followed always by adequate and supervised postural drainage.

Attention is called especially to the fact that a high percentage of patients with chronic coughs have some form of allergy. Many people are sensitive to iodine. All patients should be watched closely while this drug is being used.

Allergic cough was described by Prigal[11] as having definite characteristics. It is a loud, barking cough, paroxysmal in nature, and is relatively non-productive. It may last from a few minutes to hours or days. It defies analysis by the usual methods of examination, in that it presents negative radiographic findings in the chest, negative bacteriological findings in the sputum, and negative physical findings in the chest. The patient looks and feels well, but usually complains of an itchy, scratchy, or rubbing sensation deep in the throat.

The allergic nature of this cough is verified by certain phenomena common to all allergic diseases, such as: a history of

allergic manifestations in other members of the family, or by a history of past and/or concomitant allergic conditions, such as: urticaria, eczema, gastro-intestinal allergy, migraine, or similar conditions. There may be sensitivity to allergens, demonstrable by skin test, or by the therapeutic response to epinephrine and related drugs. Some of such patients have seasonal or perennial cough which is relieved by the removal of the causative agent, and some obtain reduction of sensitivity by hyposensitization with the allergen concerned, particularly if it is an inhalant. Antihistaminic drugs are to be used with discretion even in the allergic patient, because of the short duration of the beneficial results and because of possible reactions.

The indications for the use of x-ray therapy and the method of its administration has not changed for many years. It is usually reserved for those cases which fail to respond to other forms of treatment. Radiation to chronic infections in the sinuses and throat aid in stopping the postnasal drainage that produces a large number of chronic coughs. Mild radiation over the lungs decreases the secretion of the glands lining the bronchi and destroys a certain amount of lymphoid tissue. It is theorized that this breaking down of the lymphoid tissue liberates a protein-like substance. Relief from symptoms is slow in this type of treatment and the delay should be explained to the patient.

Personal care is important in the management of chronic coughs. Patients should be instructed to keep outside windows in sleeping quarters closed in damp or cool weather. They should avoid changes in temperature as much as possible. They should wear warm sleeping apparel at nights and warm clothing during the day. They should avoid drafts both at night and in the daytime, and should avoid dust or other irritating inhalants at all times. Exercise may well be restricted and smoking should be discontinued. If possible they should avoid others who have respiratory infections. Regular habits at all times, and a maximum amount of mental and physical rest are helpful. Every effort should be made to go into the patient's home care problems. Most patients are ignorant of hygienic care and must be taught in detail.

SUMMARY

A review of the literature has been made. These conditions have been taken up and discussed under the following headings: History, pathology, physiology, pharmacology, contagious factors, differentiation of helpful and harmful coughs. Treatment has been discussed under the heading of anodyne sedative and stimulating expectorants with a discussion of newer drugs and gases and different methods of administration. The nature and relief of allergic

cough has been discussed. X-ray therapy and its application has been discussed. The author deems the understanding of the physiology and pathology of cough more important than the other factors for the simple reason that a thorough understanding will bring about a correct diagnosis and offer greater opportunity to alleviate this symptom.

RESUMEN

Se ha hecho una revisión de la literatura. Se han tratado y discutido estas condiciones bajo los siguientes aspectos: Historia, patología, fisiología, farmacología, factores de contagio, distinción entre las toses benificas y las dañinas. Se ha discutido su tratamiento bajo el titulo de sedante anodino y expectorantes estimulantes, discutiéndose las nuevas drogas y gases y los distintos métodos de administrarlos. Se ha discutido la naturaleza de la tos alérgica y su alivio. Se ha discutido la terapia con rayos X y su aplicación. El autor considera de mayor importancia la comprensión de la fisiología y patología de la tos, que los otros factores, por la sencilla razón que su comprensión más perfecta dará lugar al diagnóstico correcto, y ofrecerá mejor oportunidad para aliviar este síntoma.

RESUME

L'auteur fait la revue de la littérature sur le problème de la toux. Les différents facteurs suivants ont été envisagés: historique, anatomie pathologique, physiologie, pharmacologie, facteurs épidémiques, différenciation des toux utiles et des toux nuisibles. L'auteur envisage le traitement sous l'angle des sédatifs sans danger, et des stimulants de l'expectoration. Il met en discussion les médications les plus nouvelles et les méthodes d'inhalation gazeuse, ainsi que leurs modes d'administration. Il étudie la nature et la façon de soulager la toux d'origine allergique. Il décrit les traitements par radiothérapie, et ses indications. L'auteur estime que les facteurs les plus importants à bien comprendre sont la physiologie et les causes anatomiques de la toux. C'est grâce à leur étude que pourra être établi un diagnostic correct et une possibilité de soulager le symptôme.

REFERENCES

1 Banyai, A. L.: "Fifteen Years' Experience with Carbon Dioxide in Management of Cough," *Dis. Chest*, 13:1, 1947.
2 Banyai, A. L. and Cadden, A. V.: "Carbon Dioxide by Inhalation in Management of Cough with Observations on Its Effects Upon Respiration," *Brit. J. Tuberc.*, 38:111, 1944.
3 Banyai, A. L.: "Modern Concept of Management of Cough (Inhalations of Oxygen and Carbon Dioxide)," *Marquette M. Rev.*, 11:1, 1945.
4 Oatway, William H. Jr.: "A Critical Analysis of Aseptic Technic for Tuberculosis," *Arizona Med.*, 1949.

5 Farber, Seymour M.: "Cough—Methods of Treatment," Personal communication, October 1948.
6 Gordonoff, T.: "Physiology and Pharmacology of Expectoration," *Ergeb. d. Physiologie*, 40:53, 1938.
7 Young, R. A.: "Modern Therapeutics: Expectorants and Linctuses," *Practitioner*, 144:433, 1940.
8 Henderson, Y.: "Acapnia as a Factor in Shock," *Brit. M. J.*, 2:1812, 1906.
"Respiratory Stimulants and Their Use," *J.A.M.A.*, 108:471, 1937.
9 Hobby, A. W.: "Recent Advancement in Treatment of Common Infections of the Lungs," *J.M.A. Ga.*, 35:350, 1947.
10 Anderson, H. H.: Personal Communication, "Drugs Affecting Respiration," Unpublished, University of Calif., September 1948.
11 Prigal, Samuel J.: "Allergic Cough," *Dis. Chest*, 8:115, 1942.

Pulmonary Aspergillosis in Greece*

PANAYOTIS CHORTIS, M.D., F.C.C.P.
Athens, Greece

Pulmonary aspergillosis is a rare disease and is confused with tuberculosis of the lungs with which it sometimes co-exists. Although numerous writers have dealt with this disease few distinct forms have been reported.

In 1943 we examined a patient who for three years had been suffering from a pulmonary disease with slight but frequent hemoptyses and progressive cachexia, with sputum negative for tubercle bacilli. We suspected mycotic infection, and from a series of repeated cultures in various media we obtained aspergillus fumigatus. We have since carried out researches for many years on a great number of patients affected with pulmonary non-tuberculous infections and detected three more cases of pulmonary aspergillosis.

Our first case was 45 years old and who for three years had been treated for bilateral fibroid tuberculosis in a sanatorium. Despite the strenuous treatment applied, not only was no improvement noticed, but on the contrary a progressive aggravation. The patient complained of excessive cough with abundant expectoration, frequent slight hemoptyses, general weakness and loss of weight. Having examined the patient three years after the onset of the disease we diagnosed fibrous lesions in upper lobes of both lungs, more especially on the right, which in fact were out of proportion to the subjective disturbances of the patient (Figure 1). What attracted our special attention was the constant negative results of the sputum examination for tubercle bacilli, as well as the abundance of whitish yellow hard granules in the sputum. Suspecting a mycotic infection we proceeded to a series of cultures of sputum and obtained several colonies of Aspergillus Fumigatus. The patient was immediately treated with large doses of calcium iodides. Due however to non-tolerance to this drug, we were compelled to change to oral administration of sulfathiazol in daily quantities of six to eight grams. This was administered with short intermissions, for four months. In all 420 grams were administered. The patient's condition started improving both in its clinical and fluoroscopic aspects after the first 20 days. At the completion of the treatment all subjective disturbances and the clinical findings had vanished, and the only trouble was certain sclerotic deposits of no clinical importance.

To make sure of complete recovery, we continued the therapy for six more months, giving for 10 days in each month, six grams of sulfathiazol per day. The patient went back to work five years ago, and has since had no complaint (Figure 2).

*Presented at the First International Congress on Diseases of the Chest, Rome, Italy, September 21, 1950.

The second case was a 50 year old civil servant, who spent his leisure time in bird and pigeon raising. From 1945 to 1947 he had complained of cough with slight mucous expectoration, evening temperatures, general weakness, and considerable loss of weight. During this time he was treated for fibroid tuberculosis of the apices of the lungs without any substantial improvement. In March 1948, the clinical picture was complicated by slight hemoptyses with abundant sputum containing necrotic elements. Two months later intense psychotic evolvements such as insomnia, nervous excitement, phobia and melancholy manifestations developed, as a result of which he was hastily taken to a neuropsychiatric clinic. After a stay there of three months there was no improvement. We were then called to examine him as he had developed hemoptysis. We excluded tuberculosis, proceeded to a sputum examination for mycosis and obtained, by culture, abundant colonies of Aspergillus Fumigatus (Figure 3).

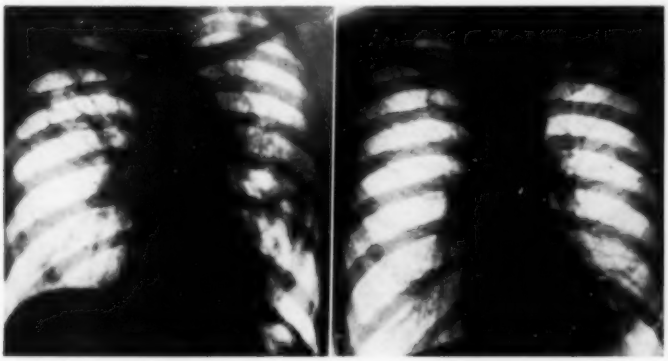


FIGURE 1 FIGURE 2

Figure 1: Lesions on both lungs, especially the left.
Figure 2: After treatment.

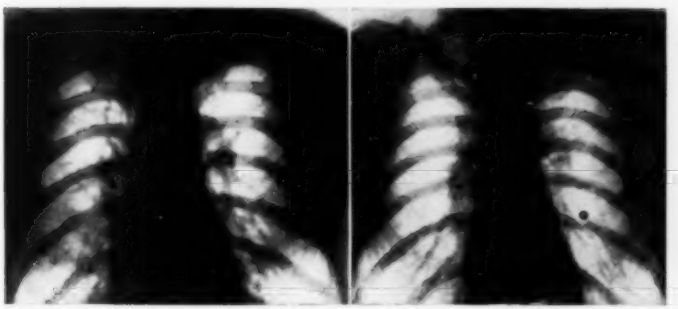


FIGURE 3 FIGURE 4

Figure 3: Infiltrative lesions. Both apices, especially the right.
Figure 4: After treatment.

Urinalysis indicated the existence of nephritis. Wasserman blood test proved negative. White cell count was 10,000. Because of the existence of nephritis, we avoided administration of sulfamides and used 800,000 units of penicillin per day during two months, with a total of 48,000,000 units. The condition of the patient started improving from the first day of the treatment, and by the end all disturbances, both clinical and neuropsychic, had entirely disappeared. Fluoroscope showed a few sclerotic evolvements, without any importance, on the apices. He returned to work and has had no complaint during the past two years (Figure 4).

The third case was a 40 year old woman, who on her farm was engaged in gathering of cereals and the raising of fowl. Four years ago she started complaining of cough with mucous expectoration and slight evening temperature elevation. Two months later the symptoms were

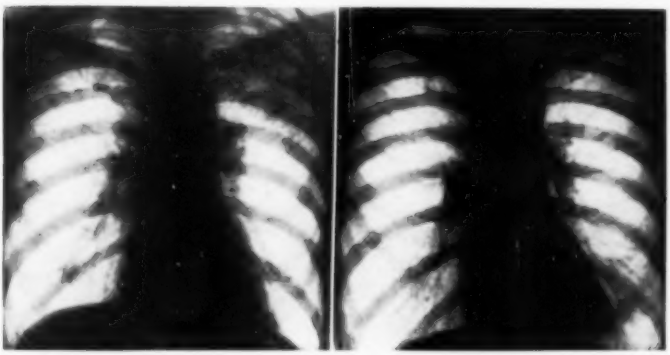


FIGURE 5 FIGURE 6

Figure 5: Infiltrative lesions. Left upper lobe, with congestion of the right upper lobe.—*Figure 6:* After treatment.

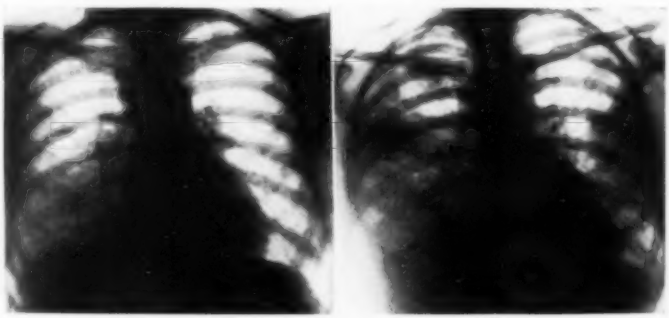


FIGURE 7 FIGURE 8

Figure 7: Extensive infiltrative lesions of the lungs, especially the right.
Figure 8: Three days before death, without treatment.

aggravated and her temperature was 38 to 39 degrees C. The attending doctor diagnosed bilateral pulmonary tuberculosis as an result of which she was sent to a sanatorium, where we discovered infiltration in the left lung with congestion of the upper right lobe (Figure 5). Tubercle bacilli were not removed from the sputum. She was steadily getting worse, her temperature remained at high levels, hemoptyses continued, expectoration increased and in it was an abundance of yellow white necrotic elements. Repeated sputum cultures revealed colonies of Aspergillus Fumigatus. Convinced as we were that, in spite of the acuteness of the course of the disease, it was a clear case of pneumonic aspergillosis, we administered 600,000 units of penicillin each 24 hours and four grams of sulfathiazole per day. This treatment was continued for 75 days during which time the patient was given 45 million units of penicillin and 300 grams of sulfathiazol. All clinical and x-ray film findings disappeared and the patient resumed her occupation. However, six months after the end of treatment, she had a relapse of the disease, for which the therapy was recommended during 15 days, and thereafter for 10 days in each of the ensuing months. Since then, and up to this day, she has had no trouble whatsoever although she resumed work two years ago (Figure 6).

The fourth case was a woman of 52 years, who on entering the hospital, presented a severe general condition with extensive bilateral pulmonary lesions (Figure 7). Bacteriological and biological sputum examinations for tubercle bacilli and the Wasserman blood test were negative. The white cell count was 8,000. Urinalysis indicated the presence of nephritis. Sputum cultures revealed colonies of Aspergillus Fumigatus. Before treatment could be administered she died of cardiac failure (Figure 8).

From the minute scrutiny of the above three cases, which following diagnosis of Aspergillosis, were successfully treated with penicillin and sulfamides, and from the pahological and anatomical research of the fourth case, it appears that the aspergillus organism, immediately after infection, locates either in the bronchial mucosa or directly in the pulmonary parenchyma. In the first case it causes an inflammatory alteration of the bronchial mucosa with involvement of the peribronchial lympho-glandular system as well as of the lymph nodules of the apices. In the second case, there resulted alveolar necrotic and inflammatory alteration. In case the necrotic alterations are predominant and are extensive, the acute form of the disease becomes pronounced, which results in the formation of cavities following the expulsion of the necrotic masses. If on the contrary the chronic inflammatory alterations are predominant, then a progressive localized or diffuse sclerosis of the pulmonary parenchyma occurs, depending on the intensity of the disease.

The main characteristic symptoms of bronchitis are persistent paroxysmal cough with mucous expectoration at the onset which gradually changes to mucopurulent, slight dyspnea and, in some

cases, paroxysms of bronchial asthma. After the extension of the disease, however, to the parenchyma, frequent hemoptyses is added and the sputum takes a characteristic appearance as it contains numerous whitish yellow elements resulting from the necrosis of the pulmonary tissue. Then follow symptoms due to the absorption of toxins and wastes of the aspergillus, which consist of anorexia, general exhaustion, loss of body weight, neuropsychic disorder, which sometimes prevail in the clinical picture, as was the case in the second instance depicted above. Finally, during the last phases, myocardial deficiency develops which conduces to a fatal issue.

Usually the course of the disease is chronic, however, some are acute.

Pulmonary aspergillosis is confused with pulmonary tuberculosis and the differential diagnosis is exceedingly difficult because both diseases have the same symptomatology and are localized in the apices of the lungs. The clinical picture of pulmonary aspergillosis is considerably milder than that of pulmonary tuberculosis, as its course is usually chronic and afebrile, affecting the patient's condition only during the final stages of the disease. On the contrary, the clinical picture in acute pulmonary aspergillosis is similar to that of tuberculosis so that the differential diagnosis can be obtained only by isolation of the pathogenetic organisms.

Sometimes pneumonic aspergillosis co-exists with pulmonary tuberculosis in which case tuberculosis prevails in the clinical picture so that the existing aspergillus organism is considered as a parasite. But even after isolation of the aspergillus, as the latter usually infects the upper respiratory tract, it is frequently difficult to set forth a rational diagnosis of aspergillosis if it is not supported by the clinical picture.

Distinct pulmonary aspergillosis appears under various forms, either as a localized fibrous nodule or in the form of diffuse infiltration mainly in the apices of the lungs, or else as a pulmonary abscess and more seldom in the cavernous form.

There are several species of Aspergilli such as the Aspergillus Nidulan, Albus Fumigatus, Helvola Niger, etc. The one most frequently found in cultures, which is pathogenic to man is the Aspergillus Fumigatus. The other species are pathogenic to plants, insects and domestic animals.

Cultivation of Aspergillus can be made on any nutrient medium, mainly on Sabouraud's anaerobic agar and on the agar broth of Czapeck. Colonies have initially a white color and with the growth of the spores the color changes to greenish, greenish-black and finally to black. Aspergilli are found in the soil, in decomposed

organic substances, in wheat, oats, bran, in the dust of dry leaves and hay, and as a parasite in the upper respiratory tract of various animals and of man. Infection takes place either through inhalation of the infected dust or by ingestion of contaminated food, or else by local inoculation in which case we have the Aspergillosis of the nose, ears, etc. The spores usually present no danger but may become pathogenic in some cases. The same applies to persons suffering from bronchitis, bronchiectasis, pulmonary tuberculosis, etc.

The disease attacks all races, and in preference male adults. Aspergillosis does not confer any immunity on man or animals.

Prognosis: Until recently the prognosis of the disease, with the exception of light localized forms with a benign course, was unfavorable. Now, with penicillin and sulfonamides, the prognosis even in severe and extensive forms is favorable, provided the diagnosis is made timely.

SUMMARY

1) Pulmonary aspergillosis is a rare disease difficult to diagnose because it is often confused with pulmonary tuberculosis.

2) In all cases of pulmonary affections where pulmonary tuberculosis is excluded, search for pulmonary aspergillosis should not be omitted.

3) The disease has usually a chronic course, but there are acute cases.

4) Pulmonary aspergillosis causes considerable harmful toxic effects on the whole organism, especially on the heart muscle (myocardium) and on the neuropsychic system.

5) Penicillin and sulfonamides whether separately administered or in combination have curative results on pulmonary aspergillosis, provided the disease is diagnosed in time.

RESUMEN

1) La aspergilosis pulmonar es una enfermedad rara, difícil de diagnosticar porque se confunde a menudo con la tuberculosis.

2) No debe omitirse la investigación de la aspergilosis en todos los casos de afecciones pulmonares cuando se ha excluído la tuberculosis.

3) La enfermedad tiene habitualmente una evolución crónica pero hay casos agudos.

4) La aspergilosis pulmonar causa considerables efectos tóxicos en el organismo especialmente en el miocardio y en el sistema neuropsíquico.

5) La penicilina y las sulfonamidas tienen efectos curativos en la aspergilosis ya se empléen solas o asociadas, siempre que el diagnóstico se haga oportunamente.

RESUME

1) L'aspergillose pulmonaire est une affection rare dont le diagnostic est difficile. Elle est habituellement confondue avec la tuberculose pulmonaire.

2) Il ne faut pas oublier de rechercher l'aspergillose pulmonaire dans les cas d'affections pulmonaires où on a pu éliminer la tuberculose.

3) L'évolution de l'affection est généralement chronique, mais il existe des formes aigues.

4) L'aspergillose pulmonaire est à l'origine d'une atteinte toxique considérable de tout l'organisme et qui frappe plus particulièrement le muscle cardiaque et le système nerveux.

5) La pénicilline et les sulfamides, soit séparément, soit associés, peuvent amener la guérison de l'aspergillose pulmonaire à condition que l'affection soit diagnostiquée suffisamment tôt.

Obscured Bronchiogenic Carcinoma
Report of a Case with Necropsy*

ISAAC EPSTEIN, M.D., F.C.C.P. and
MARJORIE J. WILLIAMS, M. B. Ch. B.
Temple, Texas

The diagnosis of bronchiogenic carcinoma is not always easy even with all modern adjuvants. Problems arise when the primary neoplasm in the bronchus is minute and cannot be recognized grossly, when the clinical and pathological picture is complicated by pre-existing chronic pulmonary infection, and when the prominent symptoms are due to metastases. The following case is reported because the diagnosis of bronchiogenic carcinoma was obscured by this triad, and the actual pathological process was only disclosed at autopsy.

History: P.L. (No. 14 825), a 50 year old Negro man, was admitted for the first time to the Veterans Administration Center, Temple, Texas, on July 1, 1949 for treatment of pulmonary tuberculosis.

He had been treated previously in eight different Veterans Administration Hospitals for arthritis and kidney disease. In 1941 he sustained a chest injury with multiple rib fractures resulting from an automobile accident. During this episode, he had chest pain, dyspnea, cough, and hemoptysis. The next year he developed bilateral pneumonia complicated by right empyema, necessitating rib resection and open drainage. According to history, he "spat" up some blood intermittently from September 1942 until May 1949, when streaking became more regular and severe.

Physical Examination: Examination on admission revealed a fairly well developed, slightly underweight, Negro man who did not appear acutely ill. The positive findings were mostly limited to the right chest where hyperresonance and markedly diminished breath sounds were present.

The x-ray film examination of the Chest (Figure 1) disclosed considerable deformity of the right thoracic cage due to old rib fractures. In the right lung field there were areas of increased radiolucency, suggesting scattered emphysematous bullae. The left lung was clear.

The laboratory findings were noncontributory. Numerous sputum examinations were all negative for acid fast organisms on smears, cultures, and guinea pig inoculation. Bronchoscopic studies on two occasions failed to reveal the source of streaking. No pathological changes were found in the bronchial tree. Bronchial aspirations were also negative for tumor cells, fungi, and tubercle bacilli.

---

*From the Departments of Thoracic Surgery and Pathology, Veterans Administration Center, Temple, Texas. Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.
Case entered in the Chest Tumor Registry, No. 2001.

The patient was discharged from the tuberculosis service on September 20, 1949 as nontuberculous and was advised to return in three months for additional studies.

On January 12, 1950 he was readmitted because of recurrence of hemoptysis during the previous two weeks. Radiological studies of the chest (Figure 2) revealed an irregular hemogenous opacity in the right infraclaviscular region which was not well seen previously. No other major changes had occurred in the lungs since the last examination on September 23, 1949. Bronchoscopy performed on January 30, 1950 showed evidence of irritation and thickening of the mucosa of the right upper lobe bronchus and blood was seen in this orifice. A biopsy, from the spur

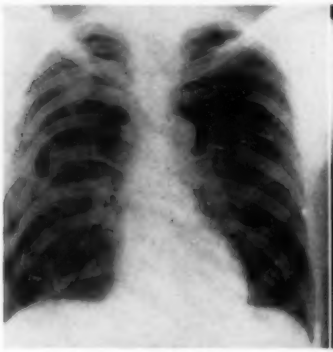


FIGURE 1

FIGURE 2

Figure 1: Roentgenogram of the chest, August 9, 1949, showing deformity of the right thoracic cage with a discrete density in the right subclavicular region.—*Fig. 2:* Note the increase in the density in the right subclavicular area.

FIGURE 3

FIGURE 4

Figure 3: Roentgenogram of the chest, February 20, 1950, following right pneumonectomy.—*Figure 4:* Note the increase in fluid in the right chest. No change in the left lung field.

of the involved bronchus, and bronchial secretion were sent to the laboratory for careful studies for malignant cells. The laboratory report again yielded negative findings. To rule out renal neoplasm with pulmonary metastases, an intravenous excretory urography done on February 2, 1950 showed no anatomical abnormalities of the kidneys.

In view of the positive radiological and bronchoscopic findings exploratory thoracotomy was performed on February 7, 1950. At exploration, all three lobes of the right lung showed evidence of severe cystic degeneration and there were areas of induration in the upper lobe. The mediastinal lymph nodes were grossly normal, and frozen section confirmed this impression. Although no definite tumor was encountered, it was considered that as the lung was obviously functionless pneumonectomy was indicated. The removed lung weighed 700 grams; chronic fibrous pleuritis, bullous emphysema, bronchiectasis and chronic pneumonitis involved all lobes. In the upper lobe there were several small necrotic areas surrounded by firm tissue. Histological examination of a number of sections confirmed the impression of a nonmalignant lesion.

Convalescence was uneventful. He was discharged on February 28, 1950 (Figure 3) with instructions to return in six weeks for upper stage thoracoplasty on the right side.

The final admission was on March 30, 1950. He had been feeling fairly well until a week prior when he experienced a sudden onset of sharp pain in the right flank and right lower abdomen which radiated to the right scrotum. There was no vomiting, nausea, or change in the bowel habits; urinary symptoms were absent.

Examination of the abdomen revealed a large mass in the right flank, aspiration of which yielded pure blood. On April 12, 1950, therefore, exploratory laparotomy was performed, and a large retroperitoneal hematoma that extended from the level of the diaphragm to the pelvis was found. Two quarts of bloody fluid were removed. The origin of the bleeding could not be found, but there was no apparent connection with the previous pneumonectomy (Figure 4). The right kidney was normal in size and consistency, and four small nodular masses were felt in the region of the adrenal gland.

Following this exploration he developed phlebothrombosis of the right iliac and femoral veins. The superficial femoral veins were ligated on April 17, 1950. His subsequent course in the hospital was downhill and he expired on May 6, 1950.

Autopsy: About 2000 cc. of serosanguineous fluid were found in the right pleural cavity; the left pleural space was normal. Careful examination of the right leaf of the diaphragm revealed no defects. The peritoneal cavity contained 1500 cc. of blood-stained fluid, and the right posterior portion of the parietal peritoneum bulged forward. Exploration of the retroperitoneal tissue in this area revealed an immense encapsulated hematoma, the boundaries of which were formed by the diaphragm and the right lobe of the liver superiorly, the duodenum and the transverse colon medially and the soft tissue of the pelvis inferiorly. Enclosed in this area were about 3000 cc. of dark blood with recent clots. In the region of the upper pole of the right kidney, there were irregular masses of friable greyish-pink tumor tissue. The right adrenal could not be identified grossly, and the thrombosed adrenal vessels terminated in the tumor mass. The right iliac vein was thrombosed throughout its entire length.

Careful examination of the right hilar structures revealed surgical occlusion of the pulmonary vessels. In the stump of the right main bronchus, there was thickening and roughening of the mucosa along its entire length. The mucosa of the trachea and the left main bronchus was hyperemic. The tracheobronchial and left bronchopulmonary lymph nodes were enlarged and grossly appeared to contain metastatic tumor. The left lung weighed 800 grams. In its lower lobe, there was some bronchiectasis and focal bronchopneumonia.

The brain weighed 1250 grams. In the anterior portion of the left parietal lobe was a well-defined hemorrhagic-like area, the greatest diameter of which was 1.8 cm.

The other visceral organs were not involved by tumor. There was a recent anemic infarct at the upper pole of the right kidney.

The significant microscopic findings were as follows: Arising in the mucosa and replacing much of the normal structures of the right bronchial stump was an undifferentiated carcinoma. The tumor cells were highly anaplastic (Figure 5). The lymphoid tissue of the tracheobronchial and bronchopulmonary nodes was extensively replaced by metastatic undifferentiated carcinoma. Sections from the brain showed that the hemorrhagic area was actually metastatic carcinoma. The right adrenal and periadrenal tissues were extensively replaced by metastatic carcinoma (Figure 6); there were also metastases in the left adrenal.

In view of the autopsy diagnosis of anaplastic bronchiogenic carcinoma arising in the stump of the right main bronchus with widespread metastases, the slides from the surgically removed right lung were re-examined. Meticulous study of these revealed small foci of carcinoma, similar to that found in the bronchus (Figures 7 and 8). The neoplastic tissue occurred predominately around blood vessels and at the margins of the areas of necrosis; occasionally tumor emboli were noted in small vessels.

FIGURE 5

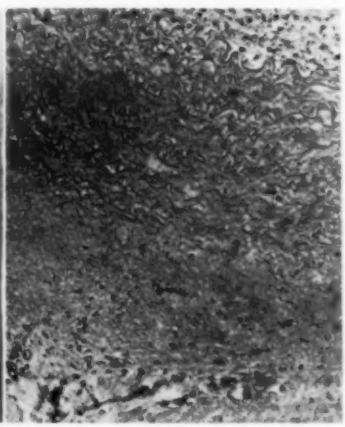


FIGURE 6

Figure 5: A low power view of the right bronchial stump showing anaplastic carcinoma arising in the mucosa; X 180.—*Figure 6:* Note the invasion of the adrenal artery by anaplastic carcinoma; X 200.

The neoplastic areas were surrounded and infiltrated by inflammatory cells. The fibro-blasts and the macrophages closely resembled the tumor cells. It must be emphasized that the tumor cells were few and that the inflammatory process was severe and extensive. It was concluded that the foci of tumor in the lung represented blood-borne metastases from the carcinoma arising in the right main bronchus.

Comment

It is well known that minute bronchiogenic carcinoma may disseminate widely producing perplexing clinical pictures. In this case we considered that an occult tumor in the right main bronchus metastasized early through the blood stream to the ipsolateral lung, and this is corroborated by the lack of involvement of the adjacent lymph nodes at the time of thoracotomy. The lung was removed distal to the tumor, which persisted in the stump.

In most autopsy series[1-7] of bronchiogenic carcinoma, the adrenals have been shown to be a favorite site for metastases. However, clinical manifestations resulting from this involvement are rare. For this reason attention is drawn to the occurrence of massive retroperitoneal hemorrhage resulting from an adrenal metastasis.

SUMMARY

1) A case of bronchiogenic carcinoma with massive retroperitoneal hemorrhage due to an adrenal metastasis is reported.

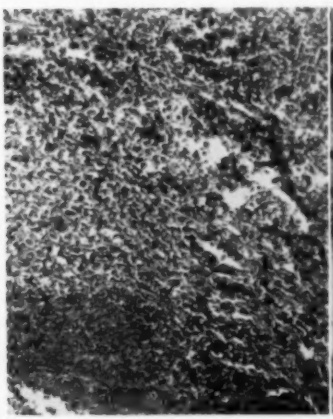


FIGURE 7

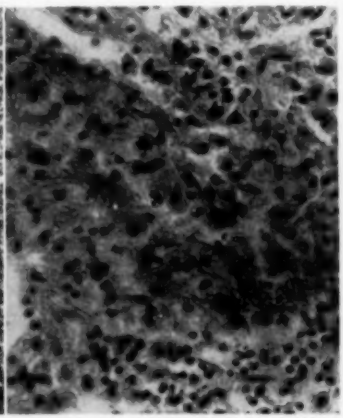


FIGURE 8

Figure 7: Section through the right lung showing a blood vessel surrounded by a zone of necrosis in the lower left corner with an area of metastatic carcinoma about it. There is chronic pneumonitis in the upper right portion; X 180.
Figure 8: High power view showing anaplastic carcinoma in the center of the field surrounded by inflammatory cells. Note the intermingling of neoplastic and inflammatory cells; X 310.

2) The ease with which small areas of blood-borne metastatic bronchiogenic carcinoma in the ipsolateral lung can be obscured by severe chronic pneumonitis is illustrated.

3) Attention is drawn to the occurrence of wide-spread metastases from minute occult bronchiogenic carcinoma.

RESUMEN

1) Se informa de un caso de carcinoma bronquiogénico con gran hemorragia retroperitoneal debida a una metástasis suprarrenal.

2) Se indica la facilidad con que pueden ser ocultadas pequeñas zones de carcinoma bronquiogénico metastásico por vía hemática, en el pulmón ipsolateral, ocultación que puede ser debida a una neumonitis crónica severa.

3) Se llama la atención al acaecimiento de metástasis diseminadas originadas por minúsculos carcinomas bronquiogénicos ocultos.

RESUME

1) Les auteurs rapportent un cas de cancer bronchique avec une hémorragie rétro-péritonéale massive, dont la cause semble être une métastase surrénale.

2) Les auteurs insistent sur la facilité avec laquelle de petites zones métastatiques de cancer bronchique peuvent être masquées dans un poumon par un tableau d'infections pulmonaires chroniques.

3) Ils attirent l'attention sur la possibilité d'ensemencement métastatique provenant de petits cancers bronchiques non décelés.

REFERENCES

1 Glomset, Daniel A.: "The Incidence of Metastasis of Malignant Tumors to the Adrenals," *Am. J. Cancer*, 32:57, 1938.

2 Jaffe, R. H.: "Primary Carcinoma of the Lung: Review of 100 Autopsies," *J. Lab. and Clin. Med.*, 20:1227, 1935.

3 Koletsky, Simon: "Primary Carcinoma of the Lung: A Clinical and Pathologic Study of One Hundred Cases," *Arch. Int. Med.*, 62:636, 1938.

4 Ochsner, A. and DeBakey, M.: "Surgical Considerations of Primary Carcinoma of the Lung: Review of Literature and Report of Nineteen Cases," *Surgery*, 8:992, 1940.

5 Olson, K. B.: "Primary Carcinoma of the Lung: A Pathological Study," *Am. J. Path.*, 11:449, 1935.

6 Reingold, Irving M., Ottoman, Richard E. and Knowaler, Benjamin E.: "Bronchiogenic Carcinoma: A Study of 60 Necropsies," *Am. J. Clin. Path.*, 20:515, 1950.

7 Willis, R. A.: "Pathology of Tumors," Butterworth, London, 1948, p. 371.

Two Cases of Intra-Bronchial Foreign Bodies of Long Duration*

JUAN M. BOETTNER, M.D., F.C.C.P., MANUEL RIVERO, M.D.,
MIGUEL A. PERITO, M.D., RAFAEL CODAS, M.D.,
NELSON LLAMOSAS, M.D. and CARLOS MAAS, M.D.
Asuncion, Paraguay

Case 1: Sara G. de R., 52 years of age, obese (89 kilos), had a chronic cough for one and one-half years which had steadily increased in severity. For some time she had not had a restful night because of violent paroxysms of cough which was productive of quantities of purulent sputum, at times streaked with blood. Periodic chills and fever, of late occurred at almost weekly intervals. There was no weight loss.

She had been treated in diverse ways, including antibiotics, with no noticeable improvement. She spontaneously tried to give her ideas as to the cause of her illness, but was interrupted by her daughter who begged her not to tell her story because she thought it would make the doctor (Dr. Boettner) laugh. However, the doctor asked the patient to tell her own story, and she explained that she was certain that her cough was due to the swallowing of a bone while eating soup.

Upon examination the patient did not appear to be acutely ill. Her temperature was 37.3 degrees C. She had hoarseness with persistent cough productive of quantities of purulent sputum. Examination of chest revealed some tenderness over the lower part of the right posterior chest wall. Breath sounds were normal throughout except for fine moist rales in the right base. Radiological inspection revealed a high right, but mobile diaphragm, and a definite, but poorly demarcated opacity in the right cardiophrenic angle. (Only after the extraction of the foreign body was it possible to detect a shadow that could correspond to it). On the first attempt to use the bronchoscope (Dr. Llamosas), a thorough examination was not possible due to the abundance of purulent sputum, but it was thought that a foreign body was seen in the inferior right bronchus. A biopsy was taken from an inflammatory nodule located in the right inferior bronchus. The pathologist reported (Dr. Carlos Maas) basal inflammatory tissue. Four days later Dr. Llamosas removed a fragment of bone 12 x 9 millimeters, perfectly preserved and without destruction of its sharp edges from the right inferior bronchus. Recovery was uneventful with rapid cessation of chronic productive cough.

Summary: (1) Chronic productive cough of one and one-half years duration following aspiration of foreign body (piece of bone). (2) Extraction of foreign body (bone) from right inferior bronchus. (3) Rapid cessation of productive cough following removal of foreign body.

Case 2: Osvaldo R., nine years of age, was first seen in the tuberculosis dispensary in October 1945, because of a chronic productive cough. He did not react to 1/1000 and 1/100 of tuberculin in December 1945, and again in 1946. He then dropped out of sight, and was not seen again until March 1949. At this time the child was emaciated, weighed 23

*From the Sanatorium "Bella Vista," Asuncion, Paraguay.

FIGURE 1

FIGURE 2

Figure 1: Fragment of foreign body (meat fibers) in right lower lobe bronchus of boy nine years of age (Case 2).—*Figure 2:* High-power view of foreign body in previous figure (striated muscle fibers well preserved after four years of lodgment in bronchus).

kilos, had clubbing of fingers and toes and chronic cough with expectoration of quantities of purulent foul smelling sputum. During the examination by Dr. Perito he spontaneously said that his cough began following the swallowing of a piece of bone while eating meat, with the production of sputum beginning some weeks later.

Examination of the chest was negative except for dullness over the right lower lobe, and numerous coarse moist rales. Radiological inspection revealed the typical picture of bronchiectasis of the basal portion of the right inferior lobe. Bronchograms were not done. He was admitted to the Sanatorium "Bella Vista" on April 4, 1949 with diagnosis of bronchiectasis for lobectomy.

Bronchoscopic examination (Dr. Llamosas), on April 4, 1949 was difficult because of abundance of purulent sputum coming from the right inferior bronchus. There was marked inflammatory reaction of the bronchial wall. Lobectomy (right inferior lobe) was accomplished on April 14, 1949 (Drs. Riveros and Codas). Operative and postoperative course were uneventful, with complete cessation of cough and production of sputum. The pathologist (Dr. Maas), reported incomplete destruction of the main bronchus of the right inferior lobe by a foreign body with sacculated and fusiform bronchiectasis. Histological study of the foreign body showed it to be made up of striated muscle fibers with their characteristic disposition. The mass was surrounded by macrophages and lymphocytic cells. In certain fields there was marked degeneration of the muscle fibers with invasion of poly-morphonuclear leukocytes.

Summary: (1) Bronchiectasis, right inferior lobe—four years duration. (2) Lobectomy, right inferior lobe with complete cure. (3) Foreign body (muscle fibers) discovered in bronchus of right inferior lobe upon examination of specimen removed at operation. Presence of foreign body establishes veracity of patient's statement that onset of illness dated back to the inhalation of a piece of meat four years ago.

Comments

The cause of diagnostic error in such cases is the *failure to think of the possible existence of a foreign body.* It is always best to allow the patient to talk of his disease even though at times his loquaciousness may distract the examiners attention. In both of the cases reported the patients knew the cause of their symptoms, but they were not heeded when they first presented themselves for treatment. In other instances *the patient is unaware of the inhalation of a foreign body.* There is cited the case of a man on board ship who one night while leaning against the rail, sneezed violently and he lost a denture which he thought fell in the water. Some hours later he developed an uncontrolled cough. Subsequent radiological inspection revealed the denture in a bronchus.

The Chevalier Jackson Clinic's ample experience in this matter give several reasons for errors in diagnosis, which are good to remember, among which are: (1) Poor anamnesis; (2) Absence of history of inhalation of foreign body; (3) Failure to consider inhalation of a foreign body; (4) Erroneous belief that foreign body is

in the digestive tract and will be expelled by normal methods; (5) Non-opaque foreign bodies.

Diagnosis: It is unnecessary to recall all the symptoms to establish the diagnosis. In the majority of cases the history is reduced to the aspiration of a foreign body, cough and suffocation. One of us (Dr. Llamosas) always does a bronchoscopy on children with history of choking, gagging, and cough. The subsequent symptoms depend on the size, shape and character of the foreign body and the point of lodgement. If there is a foreign body involved, to think of it is to make the diagnosis.

Physical Signs: Small non-destructing foreign bodies may produce no symptoms, the initial symptoms being followed by a symptomless period. If a foreign body produces partial bronchial obstruction (check valve type—Chevalier Jackson) there is often an obstructive emphysema. In complete bronchial obstruction (stop valve type—Chevalier Jackson) there is obstructive atelectasis.

Location of Foreign Body: The Chevalier Jackson Clinic upon receiving 3,112 cases of foreign bodies in the bronchi places them as follows: 36.3 per cent in the lower right bronchus; 32.7 per cent in the lower right bronchus; 89.0 per cent in the lower bronchi.

The reasons given for the selectivity of the foreign body of the lower right bronchus are: (1) Larger diameter; (2) Minimum angulation; (3) The carina is located to the left of the mid-line; (4) Greater volume of air passes through the right bronchi.

Age Groups: Of 3,112 cases of foreign bodies reported by the Chevalier Jackson Clinic, 81.6 per cent of the cases were in patients under 15 years of age.

Length of Time Foreign Body may Remain in Bronchus: Of the 3,112 cases reported by the Chevalier Jackson Clinic, 434 had had their foreign body from two months to 40 years. In the majority of these cases the foreign body was of metalic origin. Desmeules and Richard report a case where a needle had been inbedded in the wall of a bronchus 32 years.

Types of Foreign Bodies: (1) Vegetable origin. These usually produce the severest symptoms, inasmuch as they swell when moistened and give rise to complete obstruction of the bronchus. (2) Metallic objects. These are the most frequent type of foreign body. The fact that they are radio opaque aids in diagnosis. They may become corroded after a time. (3) Bones, nuts, shells, are more irritating than metal, but less than vegetable substances. They do not give rise to complete obstruction unless they are globular in shape. Chevalier Jackson reports a case where a fragment of bone was removed after 11 years. In Case 1, the fragment of bone had been present one and one-half years. (4) Meat. In the American literature reviewed, only two cases are cited, but

ptomainotoxism was not observed. In Case 2 the morsel of meat had been present four years. (5) Tonsils. Chevalier Jackson cited a case of lung abscess in a physician's child which resolved itself after the removal of a tonsil which had been aspirated at the time of a tonsillectomy six months before. (6) Other objects. Wood, glass, rubber. Undergo little alteration.

Causes of Tissue Reaction to Foreign Bodies: (1) Mechanical due to partial or complete obstruction. (2) Chemical due to actual irritation by the foreign body, especially true of vegetable seeds. (3) Allergic, especially true in vegetable seeds. (4) Infectious, foreign body actually serves as the vehicle for the entrance of bacteria, or sets up an environment favorable to the growth of bacteria.

Recent non-obstructing foreign bodies may produce no changes other than slight inflammation at the point of lodgement. However, in those of long standing obstruction nature eventually leads to complete destruction of the bronchial tissue and the formation of septic cavities. Case 2 had undergone the latter changes, and the situation is irreparable. Only with lobectomy could a cure be effected.

Pulmonary inundation (the drowned lung of Chevalier Jackson) is produced in a few days. In such cases there is abundant bronchial suppuration, and the patient literally drowns in his own pus. However, the situation is reversable by the removal of the foreign body. Case 1 is a typical example of pulmonary inundation.

Hypertrophic Pulmonary Osteo-arthropathy. (Bamberger-Marie's Disease, Hippocratic or Clubbed Fingers). The disease is probably always secondary, the majority of the cases occurring with pulmonary disease of long standing. Our case 2 demonstrated this condition.

Prognosis: The spontaneous expulsion of a foreign body from the bronchial tree does occur at times, but it cannot be depended upon. Over 98 per cent of foreign bodies can be safely removed. Peroral endoscopic removal is the only method worthy of consideration.

The pathologic changes depend upon the location and character of the object, the length of sojourn, and whether or not there is obstruction. Obstructing foreign bodies will ultimately cause suppuration and bronchiectasis. Cure of these conditions will not usually be affected by the removal of the foreign body alone. Large obstructive foreign bodies may produce asphyxiation and death.

SUMMARY

Two cases of intrabronchial foreign bodies of long duration are described. In the first case, a piece of bone remained one and one-

half years in the bronchus of the right lower lobe with symptomatology of cough, fever and suppuration. With the endoscopic extraction of the bone, rapid cessation of all symptoms was observed. The bone had not suffered any change. In the second case, a piece of meat was lodged in the bronchus of the right lower lobe for four years producing bronchiectasis of the lobe. A lobectomy cured the patient. In the histological examination, the piece of meat (muscle fibers) was found surprisingly well conserved after remaining four years in the lower bronchus.

RESUMEN

Se describen dos casos de cuerpos extraños intrabronquiales. En el primer caso, se retuvo un pedazo de hueso en el bronquio del lóbulo derecho inferior, con sintomatología de tos, fiebre y supuración. Después de efectuar la extracción endoscópica del hueso, se observó que cesaron rápidamente todos los síntomas. El hueso no había sufrido cambio alguno. En el segundo caso, se alojó un pedazo de carne en el bronquio del lóbulo derecho inferior durante cuatro años, produciéndose la bronquiectasia del lóbulo. El paciente quedó curado con una lobectomía. Al efectuarse el examen histológico, se encontró que el pedazo de carne (fibra muscular) se había conservado admirablemente bien después de permanecer cuatro años en el bronquio inferior.

RESUME

Les auteurs décrivent deux cas de corps étrangers intra-bronchiques. Dans le premier cas, un morceau d'os resta un an et demi dans la bronche lobaire inférieure droite, s'accompagnant de toux, de fièvre, et de signes de suppuration. L'extraction par bronchoscopie de l'os amena la cessation rapide de tous les symptômes. L'os n'avait subi acune altération. Dans le second cas, un morceau de viande se situait dans la bronche lobaire inférieure droite, depuis quatre ans, et avait entrainé une bronchiectasie de ce lobe. La lobectomie amena la guérison. L'examen histologique montra que le morceau de viande était admirablement conservé après être resté ainsi quatre ans dans la bronche.

REFERENCES

1 Chevalier Jackson and Chevalier L. Jackson: "Diseases of the Air and Food Passages of Foreign-body Origin," *W. S. Saunders*, 1937.

2 F. Fleischner: "Patogenia de la bronquiectasia," *Am. Rev. Tuberc.*, September 1940.

3 E. Muñoz Montt: "Enfisema pulm. parcial. Cuerpo extraño intrabronquial," *Ap. Resp. y Tub.*, Chile, 1941, Nr. 1.

4 R. Resmeules and B. Richard: "Corps etranger intrabronchique suivi de bronchiectasies et de Tub. associée," *Laval Med.*, Nov. 1942, Nr. 7. Extracted from *Am. Rev. Tuberc.*, March 1943.

5 Bunker, P. G.: "Vegetable Foreign Bodies in the Tracheobronchial Tree," *Jour. Lancet*, Nov. 1944. Extracted from *Am. Rev. Tuberc.*, Oct. 1945.

6 Chevalier Jackson and Chevalier L. Jackson: "Broncoscopia, Esofagoscopia, Gastroscopia," Trans. by P. B. Molina-Castilla, Mexico, D. F., 1945.

7 Soukup, E.: "Foreign Bodies in the Respiratory Organs," *Casop. lek. cesk.*, July 1946. Extracted from *Am. Rev. Tuberc.*, April 1947.

8 Abercromby, B. M. L.: "Foreign Bodies in the Bronchi and Oesophagus in Children," *Brit. M. J.*, Nov. 10, 1945. Extracted from *Am. Rev. Tuberc.*, April 1947.

9 Juan Max Boettner: "Anatomia y Fisiologia Bronquial," *Anales Fac. Ciencias Med.*, Asuncion, Paraguay, 1940.1. seemestre.

Spontaneous Hemopneumothorax
Report of Two Cases*

WILLIAM H. KASTL, M.D., F.A.C.S.
Alexandria, Louisiana

Spontaneous hemopneumothorax, not due to trauma, tuberculosis, or any other active pulmonary disease, is essentially a rare condition. Deiss, Gale and Brown,[1] in a recent article, found 61 reported cases to which they added two others. Subsequently, five additional cases were reported[3,4] bringing the total to 68. In this series, there were 14 deaths (20 per cent).

The pathological and clinical aspects of this condition have been adequately described by Hartzell.[2] It is accepted that the responsible mechanism is the rupture of an emphysematous bleb with the formation of a bronchopleural fistula. This may be accompanied by the tearing of one or more richly vascularized adhesions with bleeding from either the visceral or parietal sides. Symptoms arise as a result of: (1) the pulmonary collapse and mediastinal displacement due to the pressure of the blood and air, and (2) the associated blood loss.

Treatment must be directed at the restoration of the normal physiological and anatomical states. This can be accomplished by (1) removal of the accumulated blood and air from the pleural cavity with subsequent re-expansion of the lung, and (2) by restoration of the normal blood volume with whole blood transfusions. It was formerly believed that immediate removal of the blood, through lowering the intrapleural pressure, might cause increased bleeding. This concept was proved to be invalid by Sellors[6] who advocated immediate and repeated aspirations. He also emphasized the fact that since delayed aspiration of blood results in fibrin deposition on the pleura, this condition could eventually cause mediastinal fixation, fibrothorax, empyema, and inexpandable lung with depression of pulmonary function. Blood must be completely removed either by frequently repeated aspirations, or by the insertion of a catheter intercostally. This catheter should be attached to a water seal and suction system[7] with a negative pressure of 15 to 20 centimeters of water. Myers, Johnston, and

*From the Surgical Service, Veterans Administration Hospital, Alexandria, Louisiana.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Bradshaw[4] reported a case in which emergency thoracotomy was necessary because of massive, uncontrolled bleeding. If the fluid becomes loculated, infected, or if the lung fails to expand, then pulmonary decortication or even resection or thoracoplasty may become necessary. Read and Berry[5] recently reported the successful use of streptokinase-streptodornase in a case of hemopneumothorax. The action of these agents is fibrinolytic, so that they must be used with caution in the presence of a bronchopleural fistula, since, as Read warned, they can cause the reopening of an established fistula even if they cannot primarily produce one.

Once the prompt evacuation of the fluid is effected, the air is usually absorbed, allowing the lung to expand. If the bronchopleural fistula remains open, a tension pneumothorax can develop. Decompression must be obtained by the insertion of a needle or catheter intercostally, attaching it to a water seal with mild suction. However, even as low a negative pressure as 15 centimeters of water can re-open a sealed fistula, so that allowing the air to bubble into a water seal without suction may be sufficient to maintain slow expansion without air leakage. Persistent bronchopleural fistula indicates need for surgical closure.

Two cases of spontaneous hemopneumothorax are presented which we have seen on our service and which illustrate the extremes of treatment that may be necessary in the same pathological state.

Case 1 is a 27-year old white male admitted August 5, 1949, with severe pain in his left anterior hemithorax. Thirty-six hours prior to admission, he had been awakened from sleep by sharp pain in his chest. This increased in severity and he became increasingly dyspneic. There was no history of tuberculosis, but since childhood he had had occasional minor pains in the left side of his chest.

On admission, he did not appear acutely ill, but was moderately dyspneic. His temperature was 99.4 degrees Fahrenheit (orally), blood pressure was 120/70, pulse rate was 132 and respirations 32 per minute. The trachea was in the midline. There was decreased expansion, absent breath sounds and vocal fremitus over the left hemithorax. Marked hyper-resonance over the upper half and dullness to flatness over the lower half of the left chest was found. The heart sounds were muffled and rapid.

Examination of the blood revealed 9.75 grams of hemoglobin per 100 cubic centimeters, 3,400,000 erythrocytes and 17,200 leukocytes per cubic millimeter, with a differential count showing 87 per cent neutrophils, 8 per cent lymphocytes, and 4 per cent monocytes. A series of five sputum examinations and one gastric washing failed to reveal acid-fast bacilli. A roentgenogram on admission showed pneumothorax on the left side about 80 per cent complete. In addition, a fluid level was present to the tip of the third rib. The heart shadow deviated slightly toward the right.

Immediately following admission, 850 cubic centimeters of fluid were

aspirated from the left pleural cavity. This appeared to be blood but did not clot. An additional 425 cubic centimeters of the same type of fluid together with 385 cubic centimeters of air were aspirated on August 9, 1949. A total of 1000 cubic centimeters of whole blood were given by transfusion and he received 400,000 units of penicillin parenterally daily.

A low-grade fever subsided after four days. A roentgenogram on August 16, 1949, showed the left lung to be almost completely re-expanded with a minimal amount of pneumothorax at the extreme left apex. His blood count on August 17, 1949, revealed 12.8 grams of hemoglobin per 100 cubic centimeters, 4,400,000 erythrocytes and 8400 leukocytes per cubic millimeter, with a differential count of 66 per cent neutrophils and 34 per cent lymphocytes. He was discharged from the hospital August 18, 1949. A follow-up 16 months after his discharge showed that he had no pulmonary symptoms or disease since the initial episode and was in good health.

FIGURE 1

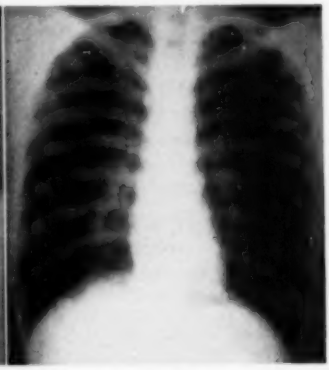


FIGURE 2

Figure 1, Case 1: Hemopneumothorax on admission.
Figure 2, Case 1: Complete expansion of lung prior to discharge.

FIGURE 3

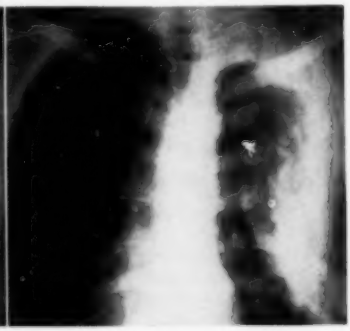


FIGURE 4

Figure 3, Case 2: Hemopneumothorax on admission.
Figure 4, Case 2: Following thoracoplasty to obliterate empyema space.

This case illustrates the ease with which hemopneumothorax may be treated providing it is seen early after onset and that the bronchopleural fistula is small and becomes sealed off well. There was no further leakage of air or blood following prompt aspiration.

Case 2 is a 32-year old white male admitted October 4, 1950, with pain in the left side of the chest and shortness of breath. On October 1, 1950, he developed sudden, sharp pain in his left hemithorax which was accompanied by marked dyspnea. This episode occurred while he was engaged in carpentry at home, under mild physical exertion. Treatment by his family physician consisted of parenteral penicillin and roentgenograms. No other therapy was received. There was no history of tuberculosis or other pulmonary disease.

On admission, the patient appeared to be acutely ill. His skin was ashen and he was moderately dyspneic. The veins of his forehead were prominent and dilated. His temperature was 98 degrees Fahrenheit (orally), his blood pressure was 110/70, pulse rate was 92 and respirations were 17 per minute. His trachea was in the midline. There was decreased expansion, absent breath sounds and vocal fremitus over the left side of his chest. Hyper-resonance over the upper half and marked dullness to flatness over the lower half of the left hemithorax were noted. There appeared to be no cardiac displacement and the heart sounds were normal.

Examination of the blood showed 9.75 grams of hemoglobin per 100 cubic centimeters, 2,840,000 erythrocytes and 8900 leukocytes per cubic millimeter, with a differential count of 77 per cent neutrophils, 19 per cent lymphocytes, and 4 per cent monocytes. His sedimentation rate was 18 millimeters per hour. A series of five sputum examinations failed to reveal acid-fast bacilli. A Mantoux test using OT Tuberculin was positive. Skin tests for histoplasmin, blastomycin and coccidioidin were negative. A roentgenogram on admission showed complete collapse of the left lung with a fluid level below the tip of the fourth rib. The mediastinal structures deviated to the right.

Because of the mediastinal shift, the extent of the pulmonary collapse, and the possibility of tension pneumothorax, decompression was effected by insertion of a catheter into the left pleural cavity through the fifth intercostal space laterally. About 1500 cubic centimeters of bloody fluid were aspirated, following which the catheter was attached to a water seal. Suction was not immediately applied to the water seal lest the bronchopleural fistula be re-opened. He received 1500 cubic centimeters of whole blood by transfusion and parenteral penicillin was given. The next day, 15 centimeters of water suction was started. A roentgenogram taken that day showed satisfactory expansion of the left lung, but with some residual mediastinal displacement and fluid at the base. From then on, expansion of the lung was slow. On October 20, 1950, two fluid pockets were evident on roentgenograms. Both presented fluid levels, one being anterior, the other posterior, at the level of the eighth thoracic vertebra. On October 23, 1950, after fluoroscopic localization, the posterior pocket was aspirated and 70 cubic centimeters of turbid, bloody fluid were removed. This fluid, on culture, was positive for staphylococcus aureus hemolyticus and was penicillin-sensitive in vitro. Re-aspiration two days later yielded 20 cubic centimeters of the same type of fluid. Subsequent roentgenograms indicated lack of further expansion and persistence of the loculated empyema. Decision to perform left pulmonary decortication

was made. At operation on November 3, 1950, many viscero-parietal adhesions were found. The lower lobe was densely adherent to the diaphragm by fibrous bands and the remainder of the lung was covered by moderately thick fibrinous peel. A small amount of fibrin was present in the pleural cavity. The bronchopleural fistula could not be found, but several small, emphysematous blebs were present on the upper lobe. These were left undisturbed. The pleural peel was removed and several resulting air leaks were sutured. Two intrapleural catheters were inserted through two intercostal spaces prior to closure. These catheters were attached to a water seal with a suction of 15 centimeters of water. His postoperative course was marked by difficulty in maintaining patency of the catheters. The lung began to re-expand slowly.

On November 20, 1950, an excessive amount of bubbling in the water seal indicated re-opening of the bronchopleural fistula. Suction was removed from the system with prompt subsidence of the bubbling and slight increase in expansion. Repeated roentgenograms failed to reveal further expansion. Therefore, on December 6, 1950, and again on December 11, 1950, streptokinase-streptodornase was instilled into the left pleural cavity through the catheter. Each time, 300 cubic centimeters of thick, plasma-like fluid were obtained, but without any appreciable lung expansion. On January 11, 1951, thoracotomy was done with re-decortication in mind. However, the lung was covered by a thick, highly vascular peel which bled excessively with the least manipulation. The fixation of the lung was such as to render consideration of lobectomy technically impossible; therefore, it was considered expedient to collapse the cavity by a modified Sauerbruch type of thoracoplasty, resecting portions of the third to eighth ribs. An intrapleural catheter was left in place and attached to a water seal. His postoperative course was smooth, with slow obliteration of the residual space about the catheter following instillations of penicillin. He was discharged from the hospital on March 14, 1951.

This case illustrates the extreme difficulty which may be encountered in the management of hemopneumothorax. The initial delay of four days prior to the first definitive treatment was instrumental in preventing complete expansion of the lung because of the formation of pleural peel. Following decortication, the difficulty in maintaining patency of the catheters again prevented re-expansion. By then, it was impossible to remove the peel without endangering the patient's condition; thus, the only alternative was thoracoplasty to obliterate the cavity. Streptokinase-streptodornase was ineffective, probably due to the excessive fibrous tissue formation.

SUMMARY

1) Two cases of spontaneous hemopneumothorax illustrate how the treatment for the same condition can vary.

2) Emphasis is placed on the prompt evacuation of blood from the pleural cavity to enhance the expansion of the lung, and the use of whole blood transfusions to combat the lowered blood volume.

RESUMEN

1) Dos casos de hemothorax espontáneo ilustran cómo el tratamiento del mismo síndrome puede variar.

2) Se hace énfasis sobre la evacuación pronta de la sangre de la cavidad pleural y el uso de transfusiones de sangre para contrarrestar la pérdida de volumen sanguíneo.

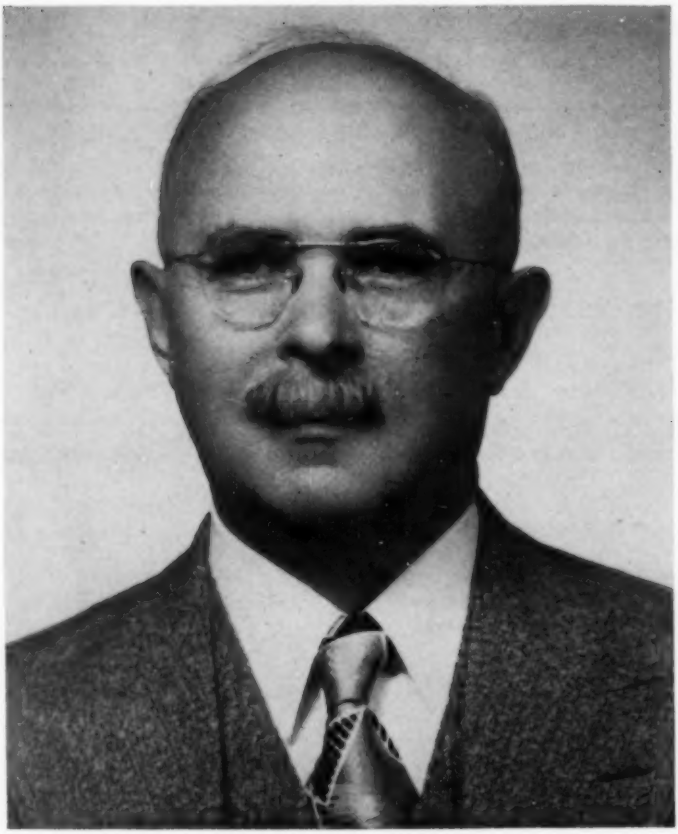
RESUME

1) Deux observations d'hémopneumothorax spontané montrent combien le traitement d'une telle affection varie selon les cas.

2) L'auteur insiste sur la nécessité d'évacuer rapidement le sang de la plèvre pour permettre la réexpansion du poumon, et sur l'utilisation de transfusions sanguines pour combattre la diminution du volume sanguin circulatoire.

REFERENCES

1 Deiss, W. P., Gale, J. W. and Brown, J. W.: "Spontaneous Hemopneumothorax. Report of Two Cases," *Am. Rev. Tuberc.*, 62:543, 1950.
2 Hartzell, H. C.: "Spontaneous Hemopneumothorax: Report of Three Cases and Review of Literature," *Ann. Int. Med.*, 17:496, 1942.
3 Moser, M.: "Spontaneous Hemopneumothorax. Treatment by Early Thoracentesis," *Dis. Chest*, 19:339, 1951.
4 Myers, R. T., Johnston, F. R. and Bradshaw, H. H.: "Spontaneous Hemopneumothcrax," *Ann. Surg.*, 133:413, 1951.
5 Read, C. T. and Berry, F. B.: "The Utilization of Streptokinase-Streptodornase," *Jour. Thoracic Surg.*, 20:384, 1950.
6 Sellors, T. H.: "Haemothorax," *Lancet*, 1:143, 1945.
7 Sweet, R. H.: "Thoracic Surgery," *W. B. Saunders Co.*, Philadelphia and London, 1950. Chapter 2, page 53.



ANDREW L. BANYAI
President

American College of Chest Physicians
1952 - 1953

Dr. Andrew L. Banyai Takes Office as College President

Andrew L. Banyai was born January 19, 1893. He graduated from the Medical School of the University of Budapest with the degree of Doctor of Medicine in 1915. He began his training in diseases of the chest shortly thereafter. Since then, he has carried on his professional activities as an internist, in private practice, in sanatorium and dispensary work.

He became Clinical Director of Muirdale Sanatorium, Milwaukee, Wisconsin, in 1928. At the same time, he joined the Medical Faculty of Marquette University, Milwaukee, Wisconsin, where at the present time he is Associate Clinical Professor of Medicine.

Doctor Banyai is a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine and was a member of the National Faculty of War-Time Graduate Meetings.

He started clinical research work in chest diseases during his early institutional affiliation. Since 1917, he has published more than ninety papers in medical journals in this country and abroad.

Doctor Banyai is recognized as the originator of artificial pneumoperitoneum treatment for pulmonary tuberculosis. This method was first used by him purposefully in 1931. His book, "Pneumoperitoneum Treatment," is a standard text in this field.

He is co-author of the book, "Fundamentals of Pulmonary Tuberculosis," and also, of the volume, "Occupational Therapy." He is Editor-in-Chief and co-author of the book, "Non-Tuberculous Diseases of the Chest," now in press.

Years ago, he popularized the topical application of cod liver oil in the treatment of tuberculous ulcers and sinuses. His roentgenologic formula for estimating the vital capacity of the lung and his studies on acroerythrosis in chest diseases attracted a great deal of attention. His pioneering work with carbon dioxide by inhalation as an expectorant is well known.

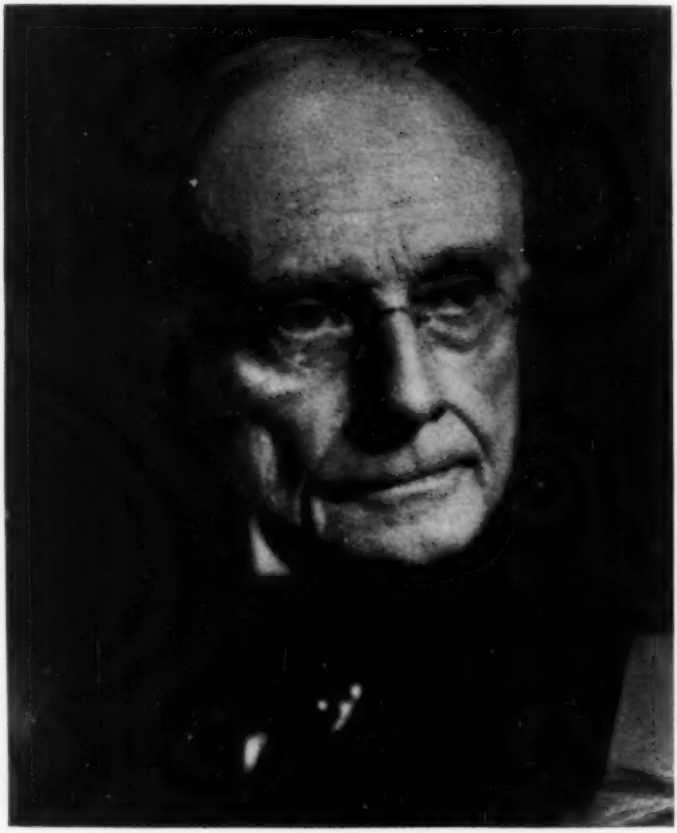
His precise, encyclopedic, authoritative lectures have made him a much-sought-for speaker at medical meetings and postgraduate courses.

Doctor Banyai is a member of the Editorial Board of "Diseases of the Chest." He is also a member of the Editorial Advisory Committee of the "American Journal of Occupational Therapy" and of the Editorial Advisory Board of GP, the official journal of the American Academy of General Practice.

Dr. Banyai has served as chairman of the Committee on International Affairs of the College and has arranged the Scientific Program for the First International Congress on Diseases of the Chest held in Rome, Italy in 1950 and for the Second International Congress on Diseases of the Chest to be held in Rio de Janeiro, Brazil in August 1952.

He has served the College in various other capacities steadfastly, with unselfish loyalty and devotion for many years.

AWARDED COLLEGE MEDAL



CHEVALIER JACKSON

Dr. Chevalier Jackson Receives College Medal

Dr. Chevalier Jackson, Philadelphia, received the Gold Medal of the American College of Chest Physicians at the President's Banquet held at the Congress Hotel, Chicago, on June 7, 1952.

Doctor Jackson was graduated from medical school in 1886 and almost immediately limited his work to laryngology. At the age of 53, upon urgent invitation, he left Pittsburgh where he was engaged in the practice of his specialty, to go to Philadelphia and establish a bronchoscopic clinic. In Philadelphia he was accorded an honor that has never been achieved by any other physician by being placed in charge of the Laryngeal Departments of the five medical schools in that city. In addition, Doctor Jackson has had many other honors bestowed upon him through a long list of honorary memberships and degrees throughout the world.

Dr. Chevalier L. Jackson, son of the recipient of the College medal, served as the president of the American College of Chest Physicians last year and received the medal for his father *in absentia*. The award was made by Dr. J. Winthrop Peabody, Washington, D. C., in the absence of Dr. H. J. Moersch, Chairman of the Committee on Award, who, due to an emergency, was obliged to return to Rochester, Minnesota.

ACCEPTANCE ADDRESS BY CHEVALIER JACKSON

(Read by Chevalier L. Jackson)

Mr. Chairman,

Members of the American College of Chest Physicians, and Honored Guests:

It is with utmost appreciation and humility, mixed with feelings of embarrassment and inferiority, that I accept this great honor.

It is embarrassing to find myself in disagreement with the unanimous decision of a committee of three of the world's highest authorities on everything related to the Chest, as a Department of Medical Science.

When I think of the great men in medicine, surgery and research who have been my predecessors as recipients of this award I get an inferiority complex.

It is my candid opinion that what little I have been able to contribute has been summarized in the verses that I wrote as a postscript to my biography, and that I venture to hope may some day be cut in a marble slab:

"Like a hick'ry cog
In the old mill wheel
He did his part
As his turn came 'round"

Eighteenth Annual Meeting of the College

A total of 932 physicians and guests attended the 18th Annual Meeting of the American College of Chest Physicians, held at the Congress Hotel, Chicago, June 5-8, 1952. The roster included members from 44 states, Hawaii, Canada and 17 other countries.

A scientific program dealing with various aspects of heart and lung disease was presented by leading physicians in the specialty. Many College members remained in Chicago to participate in the program of the Section on Diseases of the Chest of the American Medical Association.

Among the highlights of the meeting were the awarding of the College Medal to Dr. Chevalier Jackson, Philadelphia, for meritorious achievement in the specialty of diseases of the chest, and the presentation of the essay award to Dr. C. Walton Lillehei, Department of Surgery, University of Minnesota Medical School, for his essay "Experimental Bacterial Endocarditis and Proliferative Glomerulonephritis." The award included a prize of $250.

More than 500 members and guests attended the "open house" at the Executive Offices on Sunday, June 8, 1952. Refreshments were through the courtesy of the National Cylinder Gas Company of Chicago, Illinois.

Officers of the College elected to serve for 1952-53 are: President, Andrew L. Banyai, Milwaukee, Wisconsin; President-Elect, Alvis E. Greer, Houston, Texas; First Vice-President, William A. Hudson, Detroit, Michigan; Second Vice-President, James H. Stygall, Indianapolis, Indiana; Treasurer, Minas Joannides, Chicago, Illinois; Assistant Treasurer, Charles K. Petter, Waukegan, Illinois; Chairman of the Board of Regents, Donald R. McKay, Buffalo, New York; and Historian, Carl C. Aven, Atlanta, Georgia.

The 19th Annual Meeting will be held at the Hotel New Yorker, New York City, May 28-31, 1953.

Minutes of the Board of Regents Meetings

The Board of Regents of the College met in annual session at the Congress Hotel, Chicago, during the Eighteenth Annual Meeting, June 5-8, 1952. The first meeting of the Board was held at 2:00 p. m. on Thursday, June 5, and the second meeting was held on Sunday, June 8, at 4 p. m. The following Regents and other officials were present at the meetings:

James H. Stygall, Indianapolis, Indiana, Chairman, presiding
Donato G. Alarcon, Mexico City, Mexico
Robert J. Anderson, Washington, D. C.
Andrew L. Banyai, Milwaukee, Wisconsin
B. Guy Begin, Montreal, Quebec, Canada
Otto L. Bettag, Chicago, Illinois
Maurice Bonnier, Montreal, Quebec, Canada
John F. Briggs, St. Paul, Minnesota
James S. Edlin, New York, New York
Seymour M. Farber, San Francisco, California

Carl H. Gellenthien, Valmora, New Mexico
Alfred Goldman, St. Louis, Missouri
Alfred Goldman, Beverly Hills, California
Burgess L. Gordon, Philadelphia, Pennsylvania
Edward A. Greco, Portland, Maine
Alvis E. Greer, Houston, Texas
Edward W. Hayes, Monrovia, California
Paul H. Holinger, Chicago, Illinois
W. L. Howard, Northville, Michigan
Willard B. Howes, Detroit, Michigan
William A. Hudson, Detroit, Michigan
Chevalier L. Jackson, Philadelphia, Pennsylvania
Miguel Jimenez, Mexico City, Mexico
Hollis E. Johnson, Nashville, Tennessee
Harold I. Kinsey, Toronto, Ontario, Canada
Edwin R. Levine, Chicago, Illinois
Shu Fan Li, Hong Kong, China
Louis Mark, Columbus, Ohio
Amadeo V. Mastellari, Panama City, Republic of Panama
Edgar Mayer, New York, New York
Donald R. McKay, Buffalo, New York
Francisco Menendez, Havana, Cuba
Herman J. Moersch, Rochester, Minnesota
Robert B. Morrison, Austin, Texas
Jay Arthur Myers, Minneapolis, Minnesota
A. P. Naef, Lausanne, Switzerland
James M. Odell, The Dalles, Oregon
Richard H. Overholt, Brookline, Massachusetts
J. Winthrop Peabody, Washington, D. C.
Charles K. Petter, Waukegan, Illinois
Joseph C. Placak, Cleveland, Ohio
J. J. Quinlan, Kentville, N. S., Canada
William R. Rumel, Salt Lake City, Utah
Harold G. Trimble, Oakland, California
Jose F. Valiente, San Salvador, El Salvador
Wenceslao Vitug, Manila, Philippine Islands

Murray Kornfeld, Chicago, Illinois, Executive Director
Harriet Lumm Kruse, Executive Assistant
Samuel N. Turiel, Executive Assistant

Dr. Stygall called the meeting to order and introduced the guests.

A plan was presented by the Continental Casualty Company for a Group Disability Insurance Program for the benefit of College members. The purpose of the program is to make available to the membership of the College accident and sickness disability protection which provides comprehensive coverage at a premium cost below that which could be secured on an individual basis. Similar plans have been put into effect by a number of other national and international medical societies. Upon motion by Dr. Gellenthien, seconded by Dr. Jackson, the plan as proposed by the Continental Casualty Company was approved.

The following report of the Treasurer was presented by Dr. Petter:

Income:			
New Membership Fees		$22,391.00	
Dues		65,522.95	
Sales:			
Advertising	$21,560.12		
Subscriptions	9,795.96		
Directory	3,622.47		
Fellowship Keys	449.13		
Medical Books	398.90		
Color and Bleed	341.00		
	$36,167.58		
Less Discount Allowed	3,263.81	32,903.77	
Interest Received on U. S. Savings Bonds		832.50	
Postgraduate Council Income		4,047.75	
			$125,697.97
Expenses:			
Salaries		$33,202.00	
Printing of the Journal		31,291.98	
Annual Meeting		4,876.92	
U. L. A. S. T.		4,458.73	
Building Account		3,515.09	
Printing and Engraving		3,732.36	
Handling and Posting of the Journal		3,403.71	
Postage and Shipping		2,451.18	
Rent and Electricity		2,116.04	
Officer and Committee		2,084.85	
Telephone and Telegraph		2,059.32	
Office		1,831.93	
Traveling—Executive Secretary		1,494.66	
Semi-Annual Meeting, Board of Regents		1,493.24	
Public Relations		1,401.67	
Editor of the Journal		1,150.00	
Library		515.30	
Administrative Expense—Foreign Chapters		507.87	
Certificates		455.75	
Payroll Taxes		341.76	
Employment Agency Fees		304.00	
Secretary-Chairman, Board of Regents		300.00	
President's Secretarial		300.00	
Prize Essay Award		250.00	
Audit		225.00	
Insurance		173.14	
Bad Debts		155.00	
College Medal		44.48	
Contr. to Nat'l. Soc. for Med. Research		25.00	
Depreciation—Furniture and Fixtures		594.93	
TOTAL EXPENSES			$104,755.91
NET SURPLUS FOR THE YEAR			$20,942.06

The books were audited by Ralph H. Marcus of Chicago.

Dr. Myers, Chairman, Committee on Membership, reported that as of June 1, 1952 there were 4,016 members in 69 countries. The breakdown of membership according to countries was as follows:

AMERICAN COLLEGE OF CHEST PHYSICIANS

Council on Pan American Affairs (23 Countries)

Argentina	112	Jamaica	1
Bolivia	7	Mexico	64
Brazil	187	Nicaragua	1
Canada	166	Panama	13
Chile	69	Paraguay	6
Colombia	35	Peru	25
Costa Rica	6	Puerto Rico	53
Cuba	51	Trinidad	1
Dominican Republic	2	United States	2462
Ecuador	11	Uruguay	30
El Salvador	7	Venezuela	27
Guatemala	7		
Haiti	5	TOTAL	3348

Council on European Affairs (20 Countries)

Austria	3	Netherlands	5
Belgium	30	Northern Ireland	1
Czechoslovakia	1	Norway	2
England	31	Portugal	46
Finland	1	Scotland	3
France	41	Spain	21
Germany	9	Sweden	10
Greece	15	Switzerland	12
Hungary	3	Yugoslavia	1
Ireland	6		
Italy	79	TOTAL	320

Council on Pan Pacific Affairs (8 Countries)

Alaska	3	Korea	3
Guam	1	New Zealand	1
Hawaii	13	Philippines	69
Australia	34	Thailand	5
China	39		
Formosa	3	TOTAL	190
Japan	19		

Council on African and Near East Affairs (18 Countries)

Algeria	1	Lebanon	8
Belgian Congo	1	Liberia	1
Ceylon	2	Maylaya	1
Egypt	5	Morocco	1
Ethiopia	1	Pakistan	2
India	52	South Africa	45
Iran	2	Syria	1
Iraq	3	Turkey	7
Israel	23		
Jordan	2	TOTAL	158

Dr. Hayes, chairman of the Council on Undergraduate Medical Education, reported that his council had been active during the past year. A committee has been organized to work with the deans and heads of departments of the medical schools to assist them with their teaching schedules in diseases of the chest. A number of modern aids are being discussed which have been found useful in teaching programs. The details for putting this part of the program into effect are being developed.

Dr. Peabody presented the following report of the Council on Postgraduate Medical Education, of which he is chairman, listing the courses and enrollments during the past five years.

Postgraduate Course Enrollment

City	*1948*	*1949*	*1950*	*1951*	*1952*
Chicago	44	38	51	51	October 6-10
Milwaukee	—	—	—	68	41
Nashville	—	—	—	50	—
Newark	—	—	—	—	85
New York City	76	86	110	138	November 10-14
Philadelphia	62	44	47	54	87
San Francisco	43	71	—	36	43

Courses Given in Other Countries

Havana, Cuba ..... 1951 and 1952
Manila, P. I. 1951

The American College of Chest Physicians cooperated with medical schools and medical societies in presenting courses in Denver, Colorado, Houston, Texas, Minneapolis, Minnesota, and Portland, Oregon during 1951-52.

Dr. Holinger, chairman of the Committee on Motion Pictures, announced that his committee was reviewing films on diseases of the chest and that a list of the approved films, when completed, would be presented to the Board of Regents and subsequently published in the College journal. He reported that his committee is planning to assist College Chapters in obtaining films for presentation at their meetings.

A number of reports were presented by committee chairmen and referred to the interim meeting of the Board of Regents, which is to be held in Denver, Colorado, November 30, 1952, for further consideration.

Upon motion of Dr. Peabody, seconded by Dr. Hudson, Dr. Burgess L. Gordon was elected to the Committee on Nominations. Dr. Rumel, chairman of the Board of Governors, reported that Dr. T. Ashby Woodson, Louisville, Kentucky, was elected to the Committee on Nominations by the Board of Governors. The third member of the committee will be appointed by the President of the College, in accord with the College By-Laws.

Upon motion of Dr. Myers, seconded by Dr. Mark, Dr. Seymour M. Farber was elected a member of the Executive Council.

Upon motion of Dr. Hudson, seconded by Dr. Placak, Dr. Donald R. McKay was elected chairman of the Board of Regents.

The following resolutions were presented and approved by the Board:

Dr. Bettag presented a resolution, seconded by Dr. Farber, that the title of the Executive Secretary of the College be changed to Executive Director.

Resolution

WHEREAS The prime objective of the American College of Chest Physicians is to further medical education in diseases of the chest, and

WHEREAS Associate Fellows and Associate Members of the College who will eventually advance to Fellowship should be encouraged to obtain additional postgraduate study in diseases of the chest, and

WHEREAS The Council on Postgraduate Medical Education of the College presents concentrated postgraduate courses throughout the country, and

WHEREAS Members of the College should be encouraged to participate in these courses, and

WHEREAS It would be to the advantage of the Associate Fellows and Associate Members of the College to be able to enroll in brief refresher courses,

BE IT RESOLVED That in addition to the concentrated postgraduate courses sponsored by the Council, there be instructional courses especially designed for Associate Fellows and Associate Members of the College and that these courses be given in connection with the annual meetings of the College,

BE IT FURTHER RESOLVED That a system of credits be established and that such credits be accorded to members of the College enrolled in all College postgraduate courses and that these courses be recognized by the Board of Examiners,

BE IT FURTHER RESOLVED That a committee of the chairman of the Council on Postgraduate Medical Education, the chairman of the Board of Examiners and the chairman of the Board of Governors, be appointed by the President to study this phase of postgraduate medical education and to determine what significance shall attach to credits in the qualifications for fellowship and to establish the number of credits accorded for each type and each day of postgraduate instruction.

Resolution

WHEREAS The American College of Chest Physicians is now sponsoring Postgraduate Medical Education in Diseases of the Chest along several different plans and

WHEREAS The plan of organization of these courses differ in various communities

BE IT RESOLVED That the following classification for these postgraduate courses, as recommended by the Council on Postgraduate Medical Education, be adopted by the Board of Regents:

a) Those courses which for the past five years have been sponsored by the Council on Postgraduate Medical Education in large medical centers such as New York City, Chicago, and Philadelphia, shall be under the direct control and supervision of the Council.

b) Those courses which are organized on a local basis in cooperation with the American Academy of General Practice shall be under the joint sponsorship of the Council on Postgraduate Medical Education and the state chapter of the College.

c) In the event that there is more than one medical school in the city or vicinity, the Council should encourage participation of competent teachers from all medical schools in said program.

Resolution

WHEREAS A request has been received from the World Medical Association for financial assistance in helping their Association to carry out their program, and

WHEREAS The World Medical Association activities are in keeping with the expressed purposes of the American College of Chest Physicians in furthering world-wide medical education and good will among physicians throughout the world,

BE IT RESOLVED That the American College of Chest Physicians contribute a sum of $500.00 to the World Medical Association.

BE IT FURTHER RESOLVED That the American College of Chest Physicians sanction the mailing of a letter to every member of the College in the United States and Canada soliciting membership in the United States Committee of the World Medical Association.

Resolution

WHEREAS The American College of Chest Physicians has contributed the amount of twenty-five dollars ($25.00) annually to the National Society for Medical Research for the past three years, and

WHEREAS The National Society for Medical Research has indicated by letter that they will appreciate continued support from the College,

BE IT RESOLVED That a similar amount be contributed by the American College of Chest Physicians to the National Society for Medical Research for the year 1952.

Adjournment.

Report of the Committee on College Essay

The Committee on College Essay of the American College of Chest Physicians announces the winners of the 1952 Essay Contest as follows:

First Prize: C. Walton Lillehei, M.D., Department of Surgery, University of Minnesota Medical School, Minneapolis, Minnesota.

Title: "Experimental Bacterial Endocarditis and Proliferative Glomerulonephritis."

Second Prize: C. R. Woolf, M.D., London, England.

Title: "The Pathogenesis of the Normal Resolution of Acute Pneumococcal Lobar Pneumonia Treated with Sulphonamide and Penicillin; Lung Biopsy Studies."

Third Prize: Francis J. Weber, M.D., U. S. Public Health Service, Cleveland, Ohio.

Title: "Mental Hygiene in the Prevention of Irregular Discharge of Tuberculosis Patients."

Third Prize: Geoffrey L. Brinkman, M.D., Cashmere Sanatorium, Christchurch, New Zealand.

Title: "The Significance of Atelectasis in Artificial Pneumothorax."

Dr. Henry C. Sweany, Chairman of the Committee on College Essay presented the Certificate of Merit and First Prize of $250.00 to Dr. C. Walton Lillehei at the President's Banquet held at the Congress Hotel in Chicago on Saturday, June 7, 1952.

Dr. Francis J. Weber was also present to receive his Certificate of Honorable Mention. The other two certificates awarded to Dr. C. R. Woolf, London, England, and Dr. Geoffrey L. Brinkman of New Zealand, were made *in absentia*. Dr. Brinkman has arrived in the United States to accept a Fellowship at the Trudeau Sanatorium, Saranac Lake, N. Y.

Section on Diseases of the Chest American Medical Association

The Section on Diseases of the Chest of the American Medical Association elected the following officers: Joseph C. Placak, Cleveland, Ohio, Chairman; Jay Arthur Myers, Minneapolis, Minnesota, Vice-Chairman; John F. Briggs, St. Paul, Minnesota, Secretary; Hollis E. Johnson, Nashville, Tennessee, Delegate; and Edward W. Hayes, Monrovia, California, Alternate Delegate.

Edwin R. Levine, Chairman of the scientific exhibits for the Section on Diseases of the Chest, announced the following awards in this section:

The Hektoen Bronze Medal to Charles P. Bailey, Raymond C. Truez, Armand W. Angulo, Hector P. Redondo Ramirez and Nicholas Antonium of Hahnemann Medical College and Hospital, Philadelphia, and St. Michael's Hospital, Newark, New Jersey, for the exhibit on Arteriolization of the Coronary Sinus.

Certificate of Merit to Edward H. Robitzek and Irving J. Selikoff, with the assistance of Erich Mamlok and James A. Rogers, Sea View Hospital, Staten Island, New York, for the exhibit on Treatment of Tuberculosis with Hydrazine Derivatives of Isonicotinic Acid.

Certificate of Merit to Gordon M. Meade, E. Osborne Coates and William Steenken, Jr., Trudeau Sanatorium and Foundation, Trudeau, New York, for the exhibit on The Anti-Tuberculosis Activity of Two Hydrazines of Isonicotinic Acid.

Certificate of Merit to John C. Burke, Richard Donovick, Bernard S. Steinberg and Bernard Rubin, Squibb Institute for Medical Research, New Brunswick, New Jersey, for the exhibit on Isonicotinic Acid Hydrazide ("Nydrazid"): Pharmacology and Antituberculous Activity.

Certificate of Merit to DuMont F. Elmendorf, Jr., William U. Cawthon, Carl Muschenheim and Walsh McDermott, New York Hospital, New York, and Charles M. Clark, Western Navajo Hospital, Tuba City, Arizona, for the exhibit on Isonicotinic Acid Hydrazide ("Nydrazid") in the Treatment of Tuberculosis.

Honorable Mention to J. Antrim Crellin, J. Stauffer Lehman and Joseph L. Curry, Hahnemann Medical College and Hospital, Philadelphia, for the exhibit on Bronchographic Studies in Bronchiectasis Before and After Pulmonary Research.

Report of the Historian

Mr. President, Fellows of the American College of Chest Physicians, Ladies and Guests:

As we meet this evening to mark the closing of the eighteenth successful year of our College, may we pause for a few moments to pay tribute to our departed ones.

It is our sacred duty to pay honor to our heroes that died in the fight. Yes, I mean heroes that were admired and venerated for their noble deeds and qualities. In their very nature they were intrepid, brave and bold in pursuance of duties that were theirs in the course of events. These heroes of mankind are the mountains, the highlands of the moral world. Farrar once said "To live well in the quiet routine of life, to fill a little space because God wills it, to go on cheerfully with a petty round

of little duties, and little avocation, to smile for the joys of others when the heart is aching—who does this, his works will follow him. He is one of God's heroes."

This timely quotation is applicable to each of our departed conferees. Those of us that live, may we remember that there is no better armour against the shafts of death than to be busied in the service of humanity and God.

It is recorded that Aristotle when approached for giving to an unworthy person said, "I did not give it to the man, but to humanity." The gifts of these men that were placed on the alter of service were for the sake of humanity, and the promotion of our noble profession that each of us might reach the highest professional and scientific attainments. This leads to the thought that most physicians are of a rational nature that admits of nothing which is not serviceable to the rest of mankind.

To those of us that live, death will lose some of its sting if we abide by the thought in these words of Lincoln, "Let us have faith that right makes might, and in that faith, let us to the end dare to do our duty as we understand it." This span of life was lent for lofty duties, not for selfishness; not to be whiled away in aimless dreams, but to improve ourselves and mankind. Confidence in our profession leads me to believe that our departed friends have fulfilled this mission.

Herbert Acuff, Knoxville, Tennessee
Albert Meldrum Arbuckle, Pictori, Nova Scotia, Canada
Harry S. Arkin, Chicago, Illinois
Robert Graham Bell, Ottawa, Illinois
Raphael A. Bendove, New York, New York
Ludolph Brauer, Germany
Louis Cohen, Philadelphia, Pennsylvania
Seth Leroy Cox, Seattle, Washington
William G. Crawford, Terre Haute, Indiana
Gustave Derscheid, Bruxelles, Belgium
Pedro L. Farinas Mayo, Havana, Cuba
George Burton Gilbert, Colorado Springs, Colorado
Philip W. Hardie, Hamilton, Ontario, Canada
Russell Robert Hendrickson, Crookston, Minnesota
Andrew C. Henske, St. Louis, Missouri
Louis Xavier Kolipinski, Richmond, Virginia
Donald Francis MacInnis, Minneapolis, Minnesota
Francis Edward O'Brien, Haydenville, Massachusetts
Stephen Anthony Parowski, San Diego, California
Theodore Newell Rafferty, Sebring, Florida
Walter Clarence Reineking, Jacksonville, Florida
John Wallace Stacey, Tucson, Arizona
Henry Yandell Swayze, Center Point, Texas
Henry Watson, Cheltenham, Victoria, Australia
Bertha Wilensky, Toronto, Ontario, Canada

As we close the pages to the book of our own lives, may we recall these immortal words of Carlyle, "Thy life is no idle dream, but a solemn reality; it is thine own, and it is all thou hast to front eternity with."

May we accept the inheritance of our deceased ones with the faith that makes the discords of the present, the harmonies of the future.

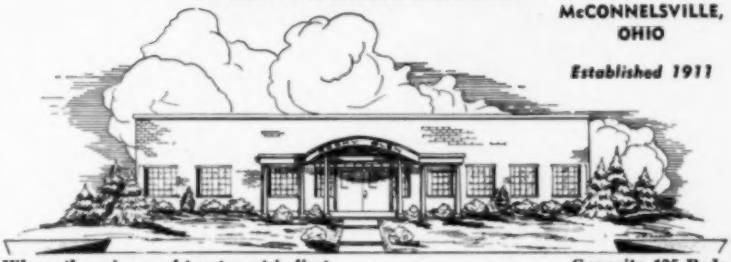
Carl C. Aven, M.D.

Rocky Glen Sanatorium

FOR TUBERCULOSIS

McCONNELSVILLE, OHIO

Established 1911



Where the science of treatment is first — *Capacity 135 Beds*

FOR THE MEDICAL AND SURGICAL TREATMENT OF TUBERCULOSIS

LOUIS MARK, M.D., Medical Director, 677 North High Street, Columbus, Ohio

HARRY MARK, Superintendent — **MRS. HARRY MARK,** Asst. Superintendent

HENRY BACHMAN, M.D., Resident Medical Director

MICHAEL L. MICHAELIS, M.D., Res. Phys. — **FELIX BACHMANN, M.D.,** Res. Phys.

L. CHANDLER ROETTIG, M.D., Surgeon — **A. N. KISHLER, D.D.S.,** Attending Dentist

Beautiful Surroundings — **Reasonable Rates**

COLLEGE EVENTS

XII Congress, International Union Against Tuberculosis
II International Congress on Diseases of the Chest
Rio de Janeiro, Brazil, August 24-30, 1952.

Intermin Session, American College of Chest Physicians,
Denver, Colorado, November 30, December 1, 1952.

19th Annual Meeting, American College of Chest Physicians,
Hotel New Yorker, New York, New York, May 28-31, 1953.

Chicago Postgraduate Course, October 6-10, 1952.

New York Postgraduate Course, November 2-6, 1952.

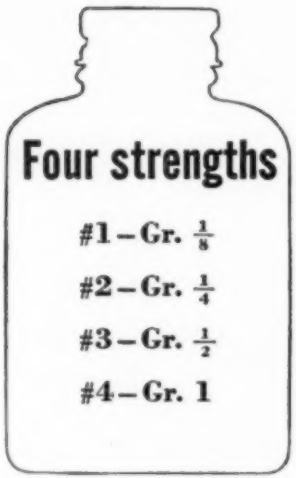
Philadelphia Postgraduate Course, March 23-27, 1953.

For Pain

'TABLOID'

'EMPIRIN' COMPOUND®

with CODEINE PHOSPHATE*



*12 times more soluble than sulfate!

BURROUGHS WELLCOME & CO. (U.S.A.) INC.
TUCKAHOE 7, N. Y.

MEDICAL SERVICE BUREAU

The facilities of the Medical Service Bureau of the American College of Chest Physicians are available to all who are interested in seeking positions or obtaining applicants in the field of chest diseases. If you wish to advertise in *Diseases of the Chest*, please write to the Medical Service Bureau, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, for rates and further information.

POSITIONS AVAILABLE

Residency available in diseases of the chest. Approved one year credit toward boards in internal medicine. Surgical training also available. Experience in all types of collapse and surgical procedures. Complete maintenance for doctor and family, situated in resort country. Must be eligible for Michigan license. Salary $4700 to $5700, depending upon experience. Please address Box 253A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Assistant physician wanted for 130 bed tuberculosis hospital in Southeast Texas. Quarters and maintenance available for single man. Can make satisfactory arrangements for married man. Salary according to experience and credentials. Please address Box 254A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Medical Director wanted for 100 bed tuberculosis hospital, North American graduate. Salary to $10,000, commensurate with qualifications and experience. Full maintenance. Please apply State Tuberculosis Hospital Commission, New State Office Bldg., Frankfort, Kentucky.

Assistant Medical Director wanted for 100 or 250 bed tuberculosis hospital, North American graduate. Salary to $9,500, commensurate with qualifications and experience. Full maintenance. Please apply State Tuberculosis Hospital Commission, New State Office Bldg., Frankfort, Ky.

Assistant Medical Director wanted with background in internal medicine for Mineral Springs Sanatorium, Cannon Falls, Minnesota; 100 bed county tuberculosis hospital with active medical, surgical and research program. Good salary plus furnished house. Man must be eligible for Minnesota license. Position opens January 1, 1953. Please address E. V. Bridge, M.D., Superintendent.

Resident physician wanted for 100 or 250 bed tuberculosis hospital. Salary to $9,000 and complete maintenance. Please apply State Tuberculosis Hospital Commission, New State Office Bldg., Frankfort, Kentucky.

Resident physician wanted for active thoracic surgery service. Salary to $9,000, commensurate with qualifications and experience. Full maintenance. Tuberculosis hospital. Please apply State Tuberculosis Hospital Commission, New State Office Bldg., Frankfort, Kentucky.

POSITION WANTED

Chest specialist, well trained and experienced. Class A school graduate. Desires position of responsibility. Outstanding references. Available immediately. Please address Box 267B, American College of Chest Physicians, 112 E. Chestnut St., Chicago 11, Illinois.

ANNOUNCEMENT

The American Trudeau Society, Medical Section of the National Tuberculosis Association, invites the submission of abstracts of scientific papers for presentation at its next annual meeting in Los Angeles, California, May 18 to 22, 1953, at the Hotel Statler.

Full information can be obtained by writing to the Chairman of the Medical Sessions Committee, American Trudeau Society, 1790 Broadway, New York 19, New York.

NEWS NOTES

The July issue of the Mississippi Medical Journal contains an article by Dr. Fred M. Meixner, a former Regent and Past President of the Illinois Chapter of the College on "What We Expect from Tuberculosis Surveys of Admissions to General Hospitals."

Dr. Aldo A. Luisada, Associate Professor of Medicine and Program Director of Cardiology of the Chicago Medical School, was cited in the June 1952 issue of the school's publication, CMS News, for his comprehensive plan of teaching and research in the study of the heart and allied fields.

In the field of health, men of different races and creeds work easily together for objectives in which all men believe and which are of benefit to all.—Frank G. Boudreau, M.D., Pub. Health Reports, April, 1952.

add weight

provide energy

MINIMIZING FERMENTATION

Anticipated effects of glucose to increase weight and provide energy are lost if the patient shows unwillingness to accept it.

When 'Dexin' is given, the common aversion to the sickly, sometimes nauseating, taste of glucose in many of its ordinary forms is noticeably absent.

'Dexin' is so palatable, so refreshing, that neither children nor adults ever need urging to take it as prescribed.

(Contains 75% Dextrins and 25% Maltose)

3-6 tablespoonfuls in milk, fruit juices, or other beverages, three or more times daily.

'Dexin'® brand

HIGH DEXTRIN CARBOHYDRATE

An improved form of glucose therapy

BURROUGHS WELLCOME & CO. (U.S.A.) INC.
Tuckahoe 7, N.Y.

ALUM ROCK SANATORIUM

SAN JOSE, CALIFORNIA

TELEPHONE CLAYBURN 8-4921

A Non-profit sanatorium for the treatment of tuberculosis and other diseases of the chest.

Visiting Medical Staff:

Harold Guyon Trimble, M.D., Oakland
Cabot Brown, M.D., San Francisco
J. Lloyd Eaton, M.D., Oakland
H. Corwin Hinshaw, M.D., San Francisco
Gerald L. Crenshaw, M.D., Oakland
Glenroy N. Pierce, M.D., San Francisco
Donald F. Rowles, M.D., Oakland
James Kieran, M.D., Oakland
Robert Stone, M.D., Oakland
William B. Leftwich, M.D., Oakland

Consulting Pathologist

E. Gwyn Roberts, M.D., San Francisco

Medical Director

Buford H. Wardrip, M.D.
Telephone Clayburn 8-4921

Associate Medical Director

C. Gerald Scarborough, M.D.

100 Beds for Crippled Children *200 Beds for Tuberculosis*

ST. JOHNS SANITARIUM, Springfield, Ill.

Complete in every detail. Rates low—because of the services of the Hospital Sisters of St. Francis.

Medical Director
DR. ROBERT K. CAMPBELL

Address
SISTER THEODINE, R.N., Supt.

Cragmor Sanatorium

For the treatment of tuberculosis and diseases of the chest, situated near Colorado Springs in the heart of the Rockies. Ideal year-round climate. Individual apartments, with or without baths. Rates on request.

For detailed information address
Henry W. Maly, M.D., Director
Cragmor Sanatorium
Colorado Springs, Colorado

SOUTHWESTERN PRESBYTERIAN SANATORIUM

ALBUQUERQUE, NEW MEXICO

★

A well-equipped Sanatorium in the Heart of the Well Country.

Write for Information and Rates

MARYKNOLL SANATORIUM

MONROVIA, CALIFORNIA

(MARYKNOLL SISTERS)

A sanatorium for the treatment of tuberculosis and other diseases of the lungs. Located in the foothills of the Sierra Madre Mountains. Southern exposure. Accommodations are private, modern and comfortable. General care of patient is conducive to mental and physical well being.

SISTER MARY PIETA, R.N.
Superintendent

E. W. HAYES, M.D.
Medical Director

SANATORIO SAN ANGEL

MEXICO CITY, D. F.

A modern Sanatorium, finest equipment, beautiful location. All types of accomodations. Well trained medical & surgical staff. Moderate rates: Dlls. 4.50 per day and up.

Institución moderna equipada para el tratamiento médico y quirúrgico de todas las afecciones respiratorias. Cuotas moderadas, desde Dlls. 4.50 por día.

Write for information to
DR. DONATO G. ALARCON
Medical Director
Amazonas Núm. 96, México City

Preparation of Manuscripts

DISEASES OF THE CHEST, the official journal of the American College of Chest Physicians, publishes manuscripts dealing with tuberculosis, non-tuberculous diseases of the chest and cardiovascular diseases. Kindly send all manuscripts to:

JAY ARTHUR MYERS, M.D., Editor-in-Chief
111 Millard Hall
University of Minnesota
Minneapolis 14, Minnesota

---

1) All manuscripts should be typewritten on white paper, 8 to 8½ by 11 inches, double or triple spaced. Tables may be single spaced, if necessary. Only one side of the paper should be used. The original copy must be submitted, and the carbon copy should be retained by the author to compare with the proofs. Manuscripts must be original, not published elsewhere, except when special permission is granted by the Editorial Board of *Diseases of the Chest*.

2) The pages should be numbered, preferably at the top right-hand corner. The name of the author should appear on each page of manuscript and on each illustration, chart and table.

3) All dates should be written as follows: *August 25, 1951*—not 8-25-51.

4) Abbreviations should not be used in the manuscript, such as R. U. L., which should be written as *right upper lobe*.

5) Illustrations should be unmounted and appropriately numbered in pencil on the back. Legends should be listed on a separate sheet at the end of the manuscript. Photographs should be black and white glossy prints, not smaller than 3 x 3 nor larger than 5 x 7 inches. Charts and graphs should be drawn on white paper with black India ink. Whenever possible, they should be made by professional medical illustrators.

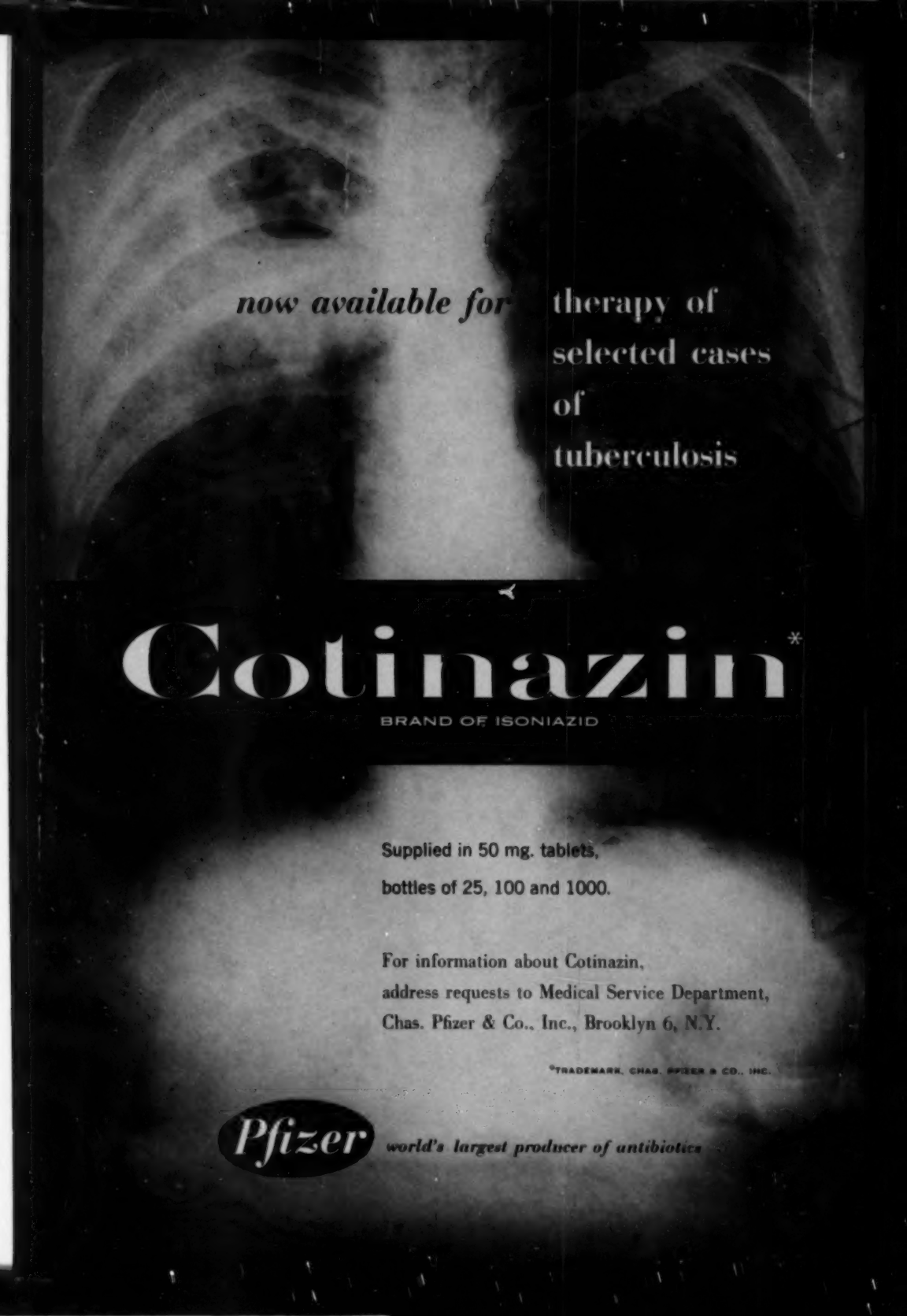
6) Written permission must accompany identifiable photographs of patients.

7) Four illustrations may be published with each article without charge. Additional photographs, when approved by the Editorial Board, may be published upon payment by the authors or the institution where the work was done.

8) Usually, long lists of references are not necessary or desirable. For most manuscripts, 10 well selected references are adequate.

9) Every paper should contain a summary in English which will be translated at the office of the Managing Editor into Spanish and French. Summaries should be brief, and contain the salient points presented in the paper in 1, 2, 3 order.

10) Authors will be given an opportunity to order reprints when they receive galley proofs for final corrections.


now available for
therapy of
selected cases
of
tuberculosis
Cotinazin*
BRAND OF ISONIAZID
Supplied in 50 mg. tablets,
bottles of 25, 100 and 1000.
For information about Cotinazin,
address requests to Medical Service Department,
Chas. Pfizer & Co., Inc., Brooklyn 6, N.Y.
*TRADEMARK, CHAS. PFIZER & CO., INC.
Pfizer
world's largest producer of antibiotics

formerly known as 'ISONIZIDE'

Microphotographs of Isoniazid 'Panray' crystals

- 50 Mg. tablets freely available on prescription and for institutional use.
- Other oral, topical and parenteral forms for clinical study.

Write for literature:

THE Panray CORP

340 Canal Street, New York 13, N. Y.

Research and Production for the Medical Profession